"HEMODYNAMIC AND TISSUE DOPPLER CORRELATES IN CONSTRICTIVE PERICARDITIS"

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CHENNAI – 600 003

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CHENNAI – 600 032

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CERTIFICATE

This is to certify that the dissertation entitled **"HEMODYNAMIC AND TISSUE DOPPLER CORRELATES IN CONSTRICTIVE PERICARDITIS"** is the bonafide original work of **DR.S.SELVAM** in partial fulfillment of the requirements for D.M. Branch-II (CARDIOLOGY) examination of THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY to be held in August 2008.The period of post-graduate study and training was from August 2005 to July 2008.

PROFESSOR R.ALAGESAN M.D., D.M.
Professor and Head of the Department of Cardiology
Madras Medical College and
Government General Hospital
Chennai – 600 003.

THE DEAN
Madras Medical College and
Government General Hospital
Chennai – 600 003.

DECLARATION

I **Dr.S.SELVAM**, solemnly declare that this dissertation entitled, **"HEMODYNAMIC AND TISSUE DOPPLER CORRELATES IN CONSTRICTIVE PERICARDITIS"** is a bonafide work done by me at the department of Cardiology, Madras Medical College and Government General Hospital during the period 2005 – 2008 under the guidance and supervision of the Professor and Head of the department of Cardiology of Madras Medical College and Government General Hospital, Professor R.Alagesan M.D.D.M. This dissertation is submitted to The Tamil Nadu Dr.M.G.R Medical University, towards partial fulfillment of requirement for the award of **D.M. Degree (Branch-II) in Cardiology.**

Place : Chennai
Date: **Dr.S.SELVAM**

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"learn to heal"

HEMODYNAMIC AND TISSUE DOPPLER CORRELATES IN CONSTRICTIVE PERICARDITIS

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INTRODUCTION

Constrictive pericarditis was recognized in the 19th century and its surgical treatment was developed early in the 20th century.
Paul Dudley White in his 1935 St Cyres lecture, described a “chronic fibrous or callous thickening of the wall of the pericardial sac that is so contracted that the normal diastolic filling of the heart is prevented . . . There may or may not be calcification . . . Parietal pericardium or epicardium may be preponderantly involved . . . one area may be involved, other areas free . . . associated heart disease is extremely rare . . . insidious evolution makes diagnosis more difficult than that of active constrictive pericarditis”. A history of several years duration and a predominant clinical feature of ascites, simulating liver disease, were notable in White's series.

Table 1(ref1) Major Historical Events in Constrictive Pericarditis

- 1669 Richard Lower describes a patient with dyspnoea and intermittent pulse
- 1842 Corrigan describes the pericardial knock (bruit de frappement)
- 1873 Kussmaul names the “ paradoxical arterial pulse,” pulsus paradoxus
- 1896 The eponym Pick’s disease is given to patients with constrictive pericarditis who have ascites and Hepatomegaly (pseudo cirrhosis).
- 1929 The first successful pericardiectomy in the United States performed by Ed Churchill.
- 1935 Paul Dudley White describes seven of 15 patients with constrictive pericarditis who were successfully operated on at the Massachussetes General Hospital
- 1946 Bloomfield demonstrates elevated right ventricular pressure with early dip and plateau in patients with constrictive pericarditis
- 1982 Isner demonstrates the value of computed tomography in the diagnosis of constrictive pericarditis

In constrictive pericarditis the easily distensible, thin, parietal and visceral pericardium linings become inflamed, thickened, and fused.
Because of these changes, the potential space between the linings is obliterated. Venous return to the heart becomes limited and ventricular filling is reduced. One of the distinguishing characteristics of this disease is the equally elevated left and right ventricular end-diastolic pressures.
Symptoms consistent with congestive heart failure (CHF), especially right-sided heart failure, develop as a result of the inability of the heart to increase stroke volume. Cardiac output gradually becomes inadequate, at first with exercise and then at rest.
Systolic function is rarely affected until late in the course of the disease, presumably secondary to infiltrative processes that affect the myocardium, atrophy, or scarring/ fibrosis of the myocardium from the overlying adjacent pericardial disease.
Haemodynamic features delineated in the 1940s and '50s included the narrow pulse pressure in the right ventricle with normal systolic pressure and greatly increased

diastolic pressure, a prominent early diastolic dip and later diastolic plateau in right ventricular pressure waveforms, and an additional prominent systolic dip in the right atrial waveform, giving a “W” atrial waveform.
Comments on the difficulty of distinguishing constrictive pericarditis from restrictive cardiomyopathy began to appear in the medical literature only after the pressure recordings from cardiac catheterization began to be used in the diagnosis of constrictive pericarditis. One may suspect that cardiac catheterization data in the two conditions were more similar than the clinical features.

In contrast to coronary artery disease, heart failure, valvular disease, and other topics in the field of cardiology, there are few data from randomized trials to guide physicians in the management of pericardial diseases. Although there are no American Heart Association/American College of Cardiology guidelines on this topic, the European Society of Cardiology has recently published useful guidelines for the diagnosis and management of pericardial diseases.
In this scenario, an attempt to investigate the hemodynamic changes in constrictive pericarditis in the era of advanced echocardiography especially Tissue doppler analysis seemed prudent in delineating this condition which mainly manifests as a pathophysiological condition

AIM OF THE STUDY

In constrictive pericarditis no physical sign or procedure is diagnostic. A constellation of clinical features along with chest radiography, echocardiography, CT scan and cardiac catheterization is often necessary in confirming the diagnosis.

Since constrictive pericarditis manifests mainly as an altered physiological state, use of advanced methods of echocardiography such as Tissue Doppler Imaging (TDI) may help in assessing and even serve as a diagnostic modality and also assess the severity of the disease in constrictive pericarditis.

With cardiac catheterization being considered as the standard in evaluating the altered hemodynamic status in constrictive pericarditis, it was considered to evaluate the role of TDI in constrictive pericarditis with cardiac catheterization data and derive possible correlates.

Hence the aim of the study is to evaluate the correlation between Tissue Doppler and hemodynamic data of cardiac catheterization study along with conventional clinical features, chest radiography, and echocardiography and CT chest analysis.

In addition, it is also the aim to delineate the extent of the disease in the study group patients during surgical management and correlate with all the available data and hence enable a complete analysis.

REVIEW OF LITERATURE

THE NORMAL PERICARDIUM

The pericardium is a relatively avascular fibrous sac that surrounds the heart. It consists of 2 layers: the visceral and parietal pericardium. The visceral pericardium is composed of a single layer of mesothelial cells that are adherent to the cardiac epicardium. The parietal pericardium is a fibrous structure that is <2 mm thick and is composed primarily of collagen and a lesser amount of elastin. The 2 layers of the pericardium are separated by a potential space that can normally contains 15 to 35 mL of serous fluid distributed mostly over the atrial-ventricular and interventricular grooves.
As a result of its relatively inelastic physical properties, the pericardium limits acute cardiac dilatation and enhances mechanical interactions of the cardiac chambers.

PATHOPHYSIOLOGY

Pericardial constriction occurs when a scarred, thickened, and frequently calcified pericardium impairs cardiac filling, limiting the total cardiac volume. The pathophysiological hallmark of pericardial constriction is equalization of the end-diastolic pressures in all 4 cardiac chambers. This occurs because the filling is determined by the limited pericardial volume, not the compliance of the chambers themselves. Initial ventricular filling occurs rapidly in early diastole as blood moves from the atria to the ventricles without much change in the total cardiac volume. However, once the pericardial constraining volume is reached, diastolic filling stops abruptly. This results in the characteristic dip and plateau of ventricular diastolic pressures. The stiff pericardium also isolates the cardiac chambers from respiratory changes in intrathoracic pressures, resulting in Kussmaul's sign.

ETIOLOGY

Pericardial constriction is usually the result of long-standing pericardial inflammation leading to pericardial scarring with thickening, fibrosis, and calcification.(2) The most frequent causes are mediastinal radiation, chronic idiopathic pericarditis, after cardiac surgery, and tuberculous pericarditis (3). The other causes are Post viral pericarditis, Chronic renal failure, Connective tissue disorders, Neoplastic pericardial infiltration, Incomplete drainage of purulent pericarditis ,Fungal and parasitic infections ,following pericarditis associated with acute myocardial infarction, following post-myocardial infarction (Dressler's) syndrome and in association with pulmonary asbestosis.

TYPES OF CONSTRICTIVE PERICARDITIS (VARIANTS OF CONSTRICTIVE PERICARDITIS)

Since 1960 the clinical profile of constrictive pericarditis has changed greatly. Tuberculous etiology has become rare in developed countries, while new etiologies have appeared. Many cases are now more appropriately considered to be acute or subacute. Subacute constrictive pericarditis differs in several respects from the chronic cases, as Paul Wood noted in his delineation of the differences between active and inactive

tuberculous constrictive pericarditis. A distinction between elastic (subacute) and rigid shell (chronic) constriction has been proposed to help to rationalize these differences (Table 3).
Other additions to the clinical profile include the recognition of effusive–constrictive pericarditis, occult constriction, localized constriction, and reversible constriction.

Table 2 Variants of constrictive pericarditis

Effusive–constrictive pericarditis	Pericardial effusion is present, sometimes loculated, with constriction by the visceral pericardium
Occult constrictive pericarditis	Haemodynamics are normal at rest, but assume the features of constriction after an acute volume load
Localised constrictive pericarditis	Constriction limited to the right or left ventricle. Ventricular interdependence reduced or absent
Transient constrictive pericarditis	During the resolution of acute pericarditis with effusion, constriction develops, but then resolves spontaneously over a few weeks

Table 3 Comparison of certain features in sub acute (elastic) and Chronic (rigid shell) constrictive pericarditis

Subacute (elastic)	*Chronic (rigid shell)*
Paradoxical pulse usually present, other signs of interdependence usually prominent	Paradoxical pulse usually minimal or absent, other signs of interdependence less prominent
Usually an XY waveform ("M" or "W" waveform)	Y is predominant, X sometimes minimal
Dip–plateau pattern less conspicuous, because early diastolic nadir may not approach zero	Dip–plateau usually conspicuous, because early diastolic nadir often reaches zero
Calcification usually absent	Calcification often present
Pericardial effusion sometimes present, generalised or loculated. Constriction is by the visceral pericardium	Pericardial effusion absent. The two layers of pericardium are fused, and jointly constrict the heart
P waves usually normal	P waves often wide, notched and low in amplitude
Atrial fibrillation or flutter rare	Atrial fibrillation or flutter common

(From: Hancock: Heart, Volume 86(3).September 1, 2001.343-349

CONSTRICTIVE PERICARDITIS WITH HISTOLOGICALLY NORMAL PERICARDIAL THICKNESS

Normal pericardial thickness is 2 mm or less, a thickness greater than 4 mm suggests pericardial constriction, and one greater than 6 mm has a high specificity for constriction. (4). Seifert et al(5) demonstrated that 12% of patients undergoing pericardial resection had a "normal" pericardium.

Causes

The most common causes are idiopathic disease (31%), cardiac surgery (25%), infection (20%), and thoracic irradiation (14%).

Symptoms

Nonspecific symptoms, including fatigue, anorexia, nausea, dyspepsia, and weight loss, were significantly more common.

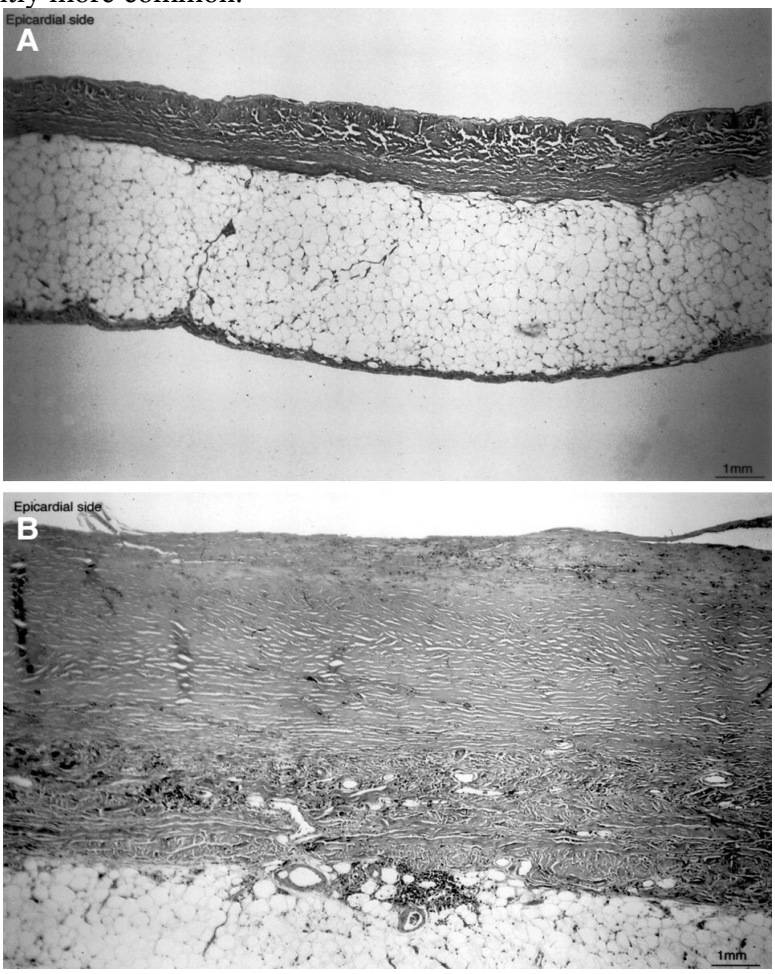


*Figure 1. A**, Pericardial specimen from patient with normal pericardial thickness**. Pericardial dimension is ≈1.5 mm in depth with 3 mm of fat on exterior surface of pericardium. B, Pericardial specimen from patient with increased pericardial thickness. Pericardial dimension is ≈7 mm in depth with fat on exterior surface of pericardium.*

PHYSICAL FINDINGS

There was little difference in the physical examination findings from the classic constrictive pericarditis , except for a greater incidence of pericardial rub (17% versus 0%; P≤0.01). No statistically significant difference was noted in the incidence of elevated jugular venous pressure or the presence of rapid X or Y descents in the jugular venous waveform or the incidence of Kussmaul sign, pulsus paradoxus, muffled heart sounds, ascites, Hepatomegaly, splenomegaly, or pleural effusion.

Pericardial thickness was normal in 18% of patients with surgically proven constrictive pericarditis. The histopathological appearance was abnormal in all patients with normal-thickness pericardial constriction, although the abnormalities were mild or focal in nature.

Thus, normal pericardial thickness does not necessarily exclude a diagnosis of constrictive pericarditis. Radical pericardiectomy is the definitive treatment.

Transient constrictive pericarditis: causes and natural history

The development of constrictive hemodynamics and subsequent resolution with medical therapy (transient CP) was first described by Sagrista-Sauleda et al. (2) in 1987 in a group of 16 patients with idiopathic pericarditis. Since then, it has been described in the setting of collagen vascular disease (6), purulent bacterial pericarditis (6,7) and chemotherapy (8).

Evidence of constrictive physiology occurring in 16 of 177 patients (9%) with effusive acute idiopathic pericarditis, with subsequent resolution with medical therapy and observation. The interval between the first echocardiogram showing PE and noninvasive recordings demonstrating CP ranged from 5 to 30 days (mean 11 days). The time to normalization of the noninvasive recordings for all patients ranged from 7 days to 58 months (mean 9.4 months). Seven patients in the study who were followed prospectively had resolution of the constrictive physiologic features after a mean of 2.7 months (range 12 days to 10 months). At a mean follow-up of 31 months, none of their patients had had a recurrence of constriction.

The authors suggested that the mechanism responsible for the findings in these patients was a transiently thickened and inelastic pericardium resulting from edema, fibrin deposition, or inflammation.

In 1993, Oh et al. (6), using Doppler echocardiography, demonstrated four instances of transient CP in conditions other than acute idiopathic pericarditis. Two were thought to be immune mediated, one was attributed to acute viral pericarditis, and one was a result of purulent bacterial pericarditis. With treatment of the underlying problem, the constrictive hemodynamics resolved in all four cases. This study extended the initial findings of Sagrista-Sauleda et al. (2) and showed that transient CP has causes other than idiopathic pericarditis

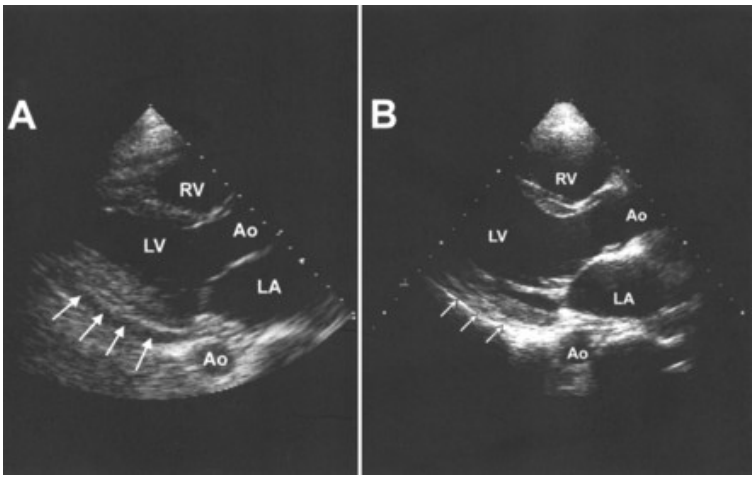


Figure 2 (A) Echocardiographic findings in constrictive pericarditis. Parasternal long-axis view of a 39-year-old man who presented one month after a motor vehicle accident with dyspnea, showing markedly increased pericardial thickness (arrows). (B) Follow-up echocardiogram performed one month later, showing near-normal pericardial thickness (arrows). Ao = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle.

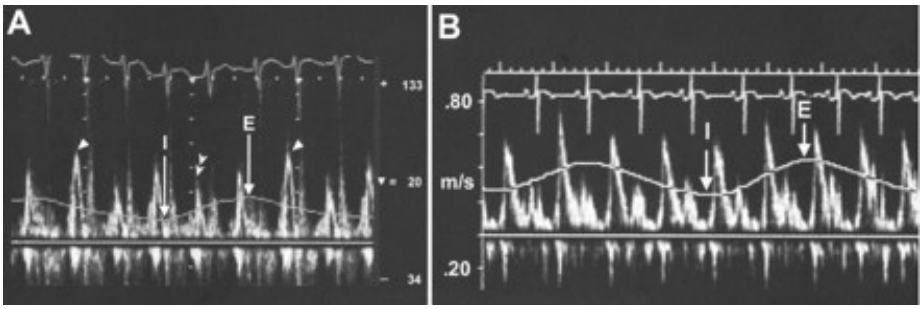


Figure 3 (A) Doppler findings in constrictive pericarditis. Pulsed-wave Doppler recording of the mitral inflow velocity showing a marked increase in the mitral inflow E velocity (single arrowhead) with expiration compared with inspiration (double arrowhead). (B) Follow-up echocardiogram one month later, showing no significant increase in the mitral inflow E velocity with expiration, consistent with resolution of constrictive hemodynamics. I = inspiration; E = expiration.

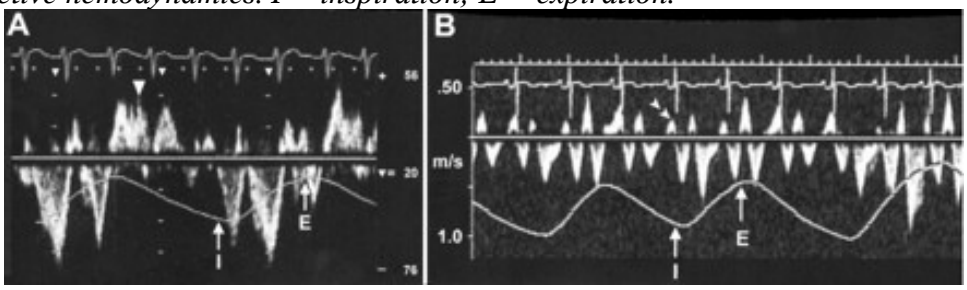


Figure 4 (A) Doppler findings in constrictive pericarditis. Pulsed-wave Doppler recording from the hepatic vein in the patient described in Figure 1, showing increased diastolic flow reversal with expiration (arrowhead). ***(B)*** *Follow-up echocardiogram performed one month later, showing an absence of increased diastolic flow reversals with expiration (double arrowhead), consistent with resolution of constrictive hemodynamics. I = inspiration; E = expiration*

Table 4: Causes of Transient Constrictive Pericarditis in 36 Patients

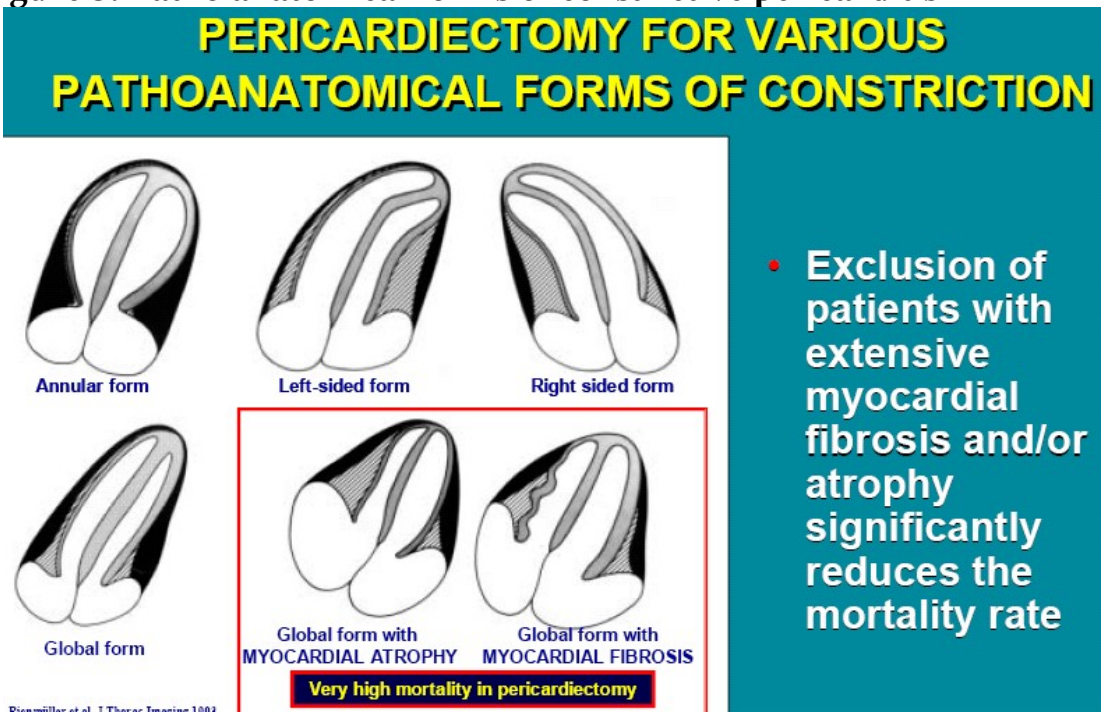
Cause	No. (%)	Resolution (Weeks)
Post pericardiectomy	9 (25)	17.4
Pericarditis, viral	7 (19)	9.8
Pericarditis, bacterial	4 (11)	18.9
Idiopathic	8 (22)	14.4
Collagen vascular disease	5 (14)	27.6
Trauma	1 (3)	16.0
Malignancy	1 (3)	8.0
Tuberculosis	1 (3)	112.0

AV GROOVE CALCIFICATION (ANNULAR CONSTRICTION)

- Occurs in calcific constrictive pericarditis; causes localized impediment to filling
- Unequal constriction of RV and LV results in hemodynamics different from classical constrictive pericarditis
- Isolated AV groove Calcification is uncommon

- May masquerade as valvular stenosis,CAD,RVOT obstruction, arrhythmias

Figure 5. Patho anatomical forms of constrictive pericarditis



(ESC Guidelines EHJ April 2004)

- Annular form –
  Bilateral thickening of the pericardium along the atrial ventricular grooves with normal configuration of both ventricles and enlargement of both atria.

- **Left sided form**
  Thickened pericardium along the left ventricle and right sided bending of the interventricular septum with tube-like configuration of mainly left ventricle and enlargement of both atria. (lateral sternotomy and partial pericardiectomy is indicated).

- **Right sided form**

  Thickened pericardium along the right ventricle and left sided bending of the interventricular septum with tube-like configuration of mainly right ventricle and enlargement of both atria (median sternotomy and partial pericardiectomy is indicated).

- **Globalform with myocardial atrophy**

  Bilateral thickening of the pericardium along both ventricles separated from the right myocardial wall by a thin layer of subepicardial fat. Tube-like configuration of both ventricles and enlargement of both atria, however, thinning of the interventricular septum and postero lateral wall of the left ventricle below 1 cm is suggesting myocardial atrophy (pericardiectomy is contraindicated).

- **Global form with peri myocardial fibrosis**

Bilateral thickening of the pericardium along both ventricles, however, the right sided thickened pericardium cannot be separated from the wave-like thin form of right sided ventricular wall suggesting perimyocardial fibrosis (pericardiectomy is contraindicated).

- **Global form**

Bilateral thickening of the pericardium along both ventricles separated from the right myocardial wall by a thin layer of subepicardial fat. Tube-like configuration of both ventricles and enlargement of both atria (median sternotomy and pericardiectomy is indicated).

Radiological classification of Constrictive Pericarditis (Reinmuller – Radiology 2003)

- Type I CP- Global pattern (47%)
  Entire pericardium is thickened or at least both ventricles and part if their corresponding atria are widely involved
- Type II CP-Focal pattern
  Localized distribution of pericardial thickening along one or both atrium
  -IIa: AV groove pericardial thickening (14%)
  - IIb: Focal involvement of ventricles (4%)
- Type III CP-Left sided pattern (1%)
  Pericardium of LV and eventually over LA is thickened
- Type IV CP- Right sided pattern (33%)
  Pericardium of RV and eventually over RA is thickened
- Type V CP- Effusive constrictive pattern (1%)
  Pericardial thickening associated with effusion

CLINICAL MANIFESTATIONS OF CLASSIC CP

Patients with pericardial constriction typically present with manifestations of elevated systemic venous pressures and low cardiac output.(9) Because there is equalization of all cardiac pressures (including right and left atrial pressures), systemic congestion is much more marked than pulmonary congestion. Typically, there will be marked jugular venous distension, hepatic congestion, ascites, and peripheral edema, while the lungs remain clear.

The limited cardiac output typically presents as exercise intolerance and may progress to cardiac cachexia with muscle wasting. In long-standing pericardial constriction, pleural effusions, ascites, and hepatic dysfunction may be prominent clinical features.(10) Patients with pericardial constriction are much more likely to have left-sided or bilateral pleural effusions than solely right-sided effusions.(11) The jugular veins are distended with prominent X and Y descents. The normal inspiratory drop in jugular venous distention may be replaced by a rise in venous pressure (Kussmaul's sign). This sign may also be present with severe right heart failure, especially in association with tricuspid regurgitation.

The classic auscultatory finding of pericardial constriction is a pericardial knock. This occurs as a high-pitched sound early in diastole when there is the sudden cessation of rapid ventricular diastolic filling.(12) When accurately recognized, a pericardial knock is a specific but insensitive indicator of pericardial constriction.

Pericardial calcification seen on the lateral plane chest x-ray is suggestive of pericardial constriction(13) Similarly, most patients with pericardial constriction have a thickened

pericardium (>2 mm) that can be imaged by echocardiography, CT, and MRI (Figure 6). (10,14,15) It is important to recognize, however, that pericardial constriction can be present without pericardial calcium and, in some cases, even without pericardial thickening. For example, in a series of 143 patients from the Mayo Clinic with surgically proven pericardial constriction, 26 (18%) had a normal pericardial thickness (<2 mm).(16) Finally, the pericardial constriction may be predominantly localized to one region of the heart.

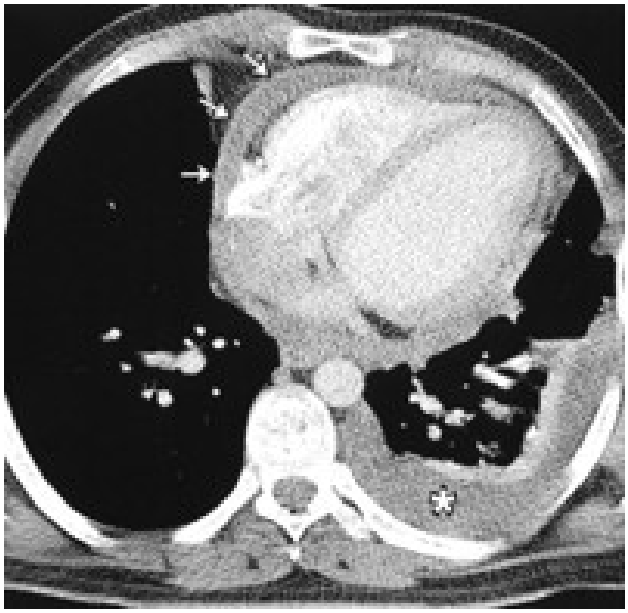


Figure 6. Chest CT from a patient with pericardial constriction showing thickened pericardium (arrows) and a left pleural effusion

Doppler echocardiography is important in the evaluation of patients with suspected pericardial constriction. The echocardiogram may demonstrate pericardial thickening and calcification. However, increased pericardial thickness can be missed on a transthoracic echocardiogram. Transesophageal echocardiography is more sensitive and accurate in determining pericardial thickness. (17) Transesophageal echocardiography can also assess pulmonary venous flow.

Doppler echocardiography frequently demonstrates restricted filling of both ventricles with a rapid deceleration of the early diastolic mitral inflow velocity (E wave) and small or absent A wave. In addition, there is substantial (>25%) respiratory variation of the mitral inflow velocity (18) .Wide swings in the E wave velocity may also occur in patients with respiratory disease, but these are associated with marked respiratory variation in the superior vena caval flow velocity (typically >20 cm/s), whereas the variation with pericardial constriction is less.(19,20) Other findings in constrictive pericarditis include preserved diastolic mitral annular velocity, rapid diastolic flow propagation to the apex, and diastolic mitral regurgitation.(21)

DIFFERENTIALDIAGNOSIS

Pericardial constriction should be considered in any patient with unexplained systemic venous congestion. Echocardiography is useful in differentiating pericardial constriction from right heart failure due to tricuspid valve disease and/or associated pulmonary hypertension.

The most difficult differentiation is between pericardial constriction and restrictive cardiomyopathy (Table 5). Clinical manifestations of restrictive cardiomyopathy most typically due to cardiac amyloid may be very similar to those due to pericardial constriction. Doppler echocardiography is the most useful method to distinguish constriction from restriction. Patients with pericardial constriction have marked respiratory variation (>25%) of mitral inflow, whereas this is not present in restrictive cardiomyopathies.
In some cases of pericardial constriction with markedly elevated venous pressures, the respiratory variation may only be present after head-up tilt. The tissue Doppler measurement of mitral annular velocities is useful in distinguishing constriction from restriction. The early diastolic mitral annular velocity (Ea) is almost always reduced in patients with myocardial

Table 5 ***Differentiation of Pericardial Constriction from Restrictive Cardiomyopathy***

	Pericardial Constriction	***Restrictive Cardiomyopathy***
Physical examination		
Pulmonary congestion	Usually absent	Usually present
Jugular venous pulse	Prominent Y descent	
Early diastolic sound	Pericardial knock	S3 (low pitched)
Pericardial thickness	>2 mm (but <2 mm in 15%)	<2 mm
Echo/Doppler findings		
LV myocardium	Normal	"Sparkling" myocardium in amyloidal
Atrial size	+/– Atrial enlargement	Atrial enlargement
Mitral valve flow pattern	Restricted	Restricted
Respiratory variation in E wave	>25%	<20%
Mitral annular diastolic velocity	>8 cm/s	<8 cm/s
Biomarker		
B-type natriuretic peptide	<200 pg/mL	>600 pg/mL
Hemodynamics		
Y descent	Prominent	Variable
PA systolic pressure	<50 mm Hg	>60 mm Hg
PCW-RA pressure	0	5 mm Hg
Reciprocal respiratory variation in RVSP/LVSP	Present	Absent

restriction, whereas it remains normal in patients with pericardial constriction. The optimal discrimination occurs with an Ea velocity of 8 cm/s. Similarly, rapid propagation

of early diastolic flow to the apex is preserved in constriction and reduced in restriction. A slope 100 cm/s of the first aliasing contour in the color M-mode best distinguishes the two. (21)

It has recently been reported that patients with pericardial constriction have only minimally elevated B-type natriuretic peptide (<200 pg/mL), whereas the B-type natriuretic peptide levels are typically markedly increased in patients with restrictive cardiomyopathy (>600 pg/mL).(22)

Traditionally, constriction and restriction were differentiated at cardiac catheterization by hemodynamic criteria. In constriction, there is a usually almost exact equalization of late diastolic pressure in both the right and left heart. With restriction, typically left ventricular end-diastolic pressure exceeds right ventricular pressure by at least a few mm Hg. Pulmonary hypertension is frequently seen with restriction but is not typically present with constriction. Thus, right ventricular diastolic pressure should be more than one third of the right ventricular systolic pressure in pericardial constriction.

It should be recognized that the aforementioned classic hemodynamic criteria have limited specificity (24% to 57%) in distinguishing pericardial constriction from cardiomyopathies.(23)

In contrast, dynamic respiratory variations indicating increased ventricular interdependence are superior. In constriction during inspiration, right ventricular systolic pressures increase, while left ventricular systolic pressure decreases. The inverse occurs during expiration. This finding had >90% sensitivity and specificity in recognizing constrictive pericarditis versus restriction in a series of 36 patients from the Mayo Clinic. (23)

Endomyocardial biopsy performed during catheterization can also be utilized in selected cases to distinguish myocardial disease from pericardial constriction.(24)

Bush et al(25) first observed that, in some patients, the hemodynamic findings of constriction may only be present after rapid volume loading and labeled this syndrome occult constrictive pericarditis. Some patients with this syndrome may improve after removal of the pericardium. The sensitivity and specificity of the response to volume loading and the role of pericardiectomy in treating this condition are not well established. (26)

Treatment

In some patients with relatively acute onset pericardial constriction, the symptoms and constrictive features may resolve with medical therapy alone.(27) For example, Haley et al(28) reported a series of 36 patients with pericardial constriction that resolved with treatment with the use of anti-inflammatory agents, colchicine, and/or steroids.

In more chronic pericardial constriction, definitive treatment is surgical pericardial decortication, widely resecting both the visceral and parietal pericardium.(10) This operation is a major undertaking with substantial risk (>6% mortality even in the most experienced centers).(29,9) The largest surgical series from the Mayo Clinic and the Cleveland Clinic indicate that patients with constriction due to idiopathic or viral pericarditis do best and patients with radiation-induced constriction fare most poorly after surgery.(29,9)

EFFUSIVE CONSTRICTIVE PERICARDITIS

Hancock (30) first recognized that some patients presenting with cardiac tamponade did

not have resolution of their elevated right atrial pressure after removal of the pericardial fluid. In these patients, pericardiocentesis converted the hemodynamics from those typical of tamponade to those of constriction. Thus, the restriction of cardiac filling was not only due to the pericardial effusion but also resulted from pericardial constriction (predominantly the visceral pericardium).

Sagrista-Sauleda et (26)recently reported a consecutive series of >1000 patients with pericarditis, 218 of whom had cardiac tamponade and underwent pericardiocentesis. In 15 of these patients, the right atrial and right ventricular diastolic pressures remained elevated with a dip and plateau morphology after the pericardiocentesis, and thus they were considered to have effusive constrictive pericarditis. The most common cause was idiopathic pericarditis as well as malignancies and after radiation. One patient had tuberculous pericarditis. Effusive constrictive pericarditis most likely represents an intermediate transition from acute pericarditis with pericardial effusion to pericardial constriction.(31)

CHEST RADIOGRAPHY

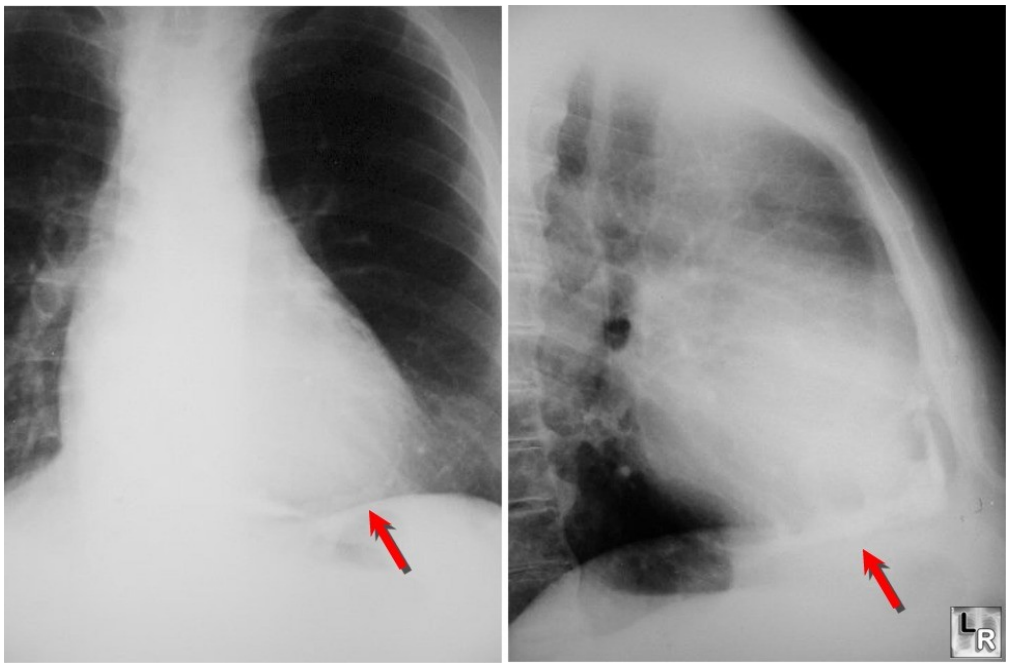


Figure 7 PA and lateral close-ups show thick pericardial calcification around apex of heart from patient with history of tuberculous pericarditis

Table 7 **Distribution of Pericardial Calcifications in Constrictive Pericarditis**

Location	Patients, *n* (%)
Inferior	33 (97)
Anterior (right ventricle)	26 (76)
Left atrioventricular groove	21 (62)
Right ventricular outflow	19 (56)
Base of left ventricle	19 (56)
Right atrioventricular groove	17 (50)
Right atrium	9 (26)
Left ventricular apex	8 (24)
Left atrium	1 (3)

* For 2 patients, radiographs were not available for review.

Calcification of the pericardium is most likely inflammatory in nature and can be seen with a variety of infections, trauma, and neoplasms. Calcification most commonly occurs along the inferior diaphragmatic surface of the pericardium surrounding the ventricles. Thin, egg-shell like calcification is more often associated with viral infection or uremia. Calcification from old TB is often thick, confluent, and irregular in appearance, especially when compared with myocardial calcification.
Pericardial calcification is not specific for constrictive pericarditis

- A calcified pericardium is not necessarily a constricted one
- Lateral chest film is useful for its detection in the atrioventricular groove or along the anterior and diaphragmatic surfaces of the right ventricle.
- Pleural effusions are present in about 60 % of patients
- Persistent unexplained pleural effusions can be the presenting manifestation
- CT or MRI are superior in the assessment of pericardial anatomy and thickness

MAGNETIC RESONANCE AND COMPUTED TOMOGRAPHIC IMAGING

CT and MRI of the thorax have been used since the early 1980s as an improved method of evaluating abnormal thickening of the pericardium. Most cases of constrictive pericarditis do indeed show an apparent pericardial thickness of 3 mm or more, at least in some areas.
CT and MRI often appear to show only focal areas of pericardial thickening in cases where the constriction is present around the entire heart. Surgeons often note variable degrees of pericardial thickness in different areas that do not necessarily correspond to differences in the degree of constriction. It is perhaps insufficiently realized, however, that some patients have constriction with relatively small degrees of thickening.
The normal pericardium is less than 1.0 mm thick; a considerable increase may not exceed the threshold of abnormality in a CT or MRI. Indeed, the constricting pericardium can be visually unimpressive, or even appear normal at first glance to the surgeon at the time of operation. Some cases of occult constriction appear to have anatomically normal visceral and parietal pericardium.
Thus, the principal limitation of CT and MRI is the occurrence of falsely negative studies. In addition, the finding of thickened pericardium does not necessarily indicate that constriction is present.
CT and MRI have approximately equal value in demonstrating thickening of the

pericardium. CT is therefore preferable in most cases, with MRI usually reserved for patients with an intolerance of iodinated contrast agent. CT is analogous to a single "snapshot", while MRI represents the average of many heart beats, gated to the cardiac cycle.(59-66)

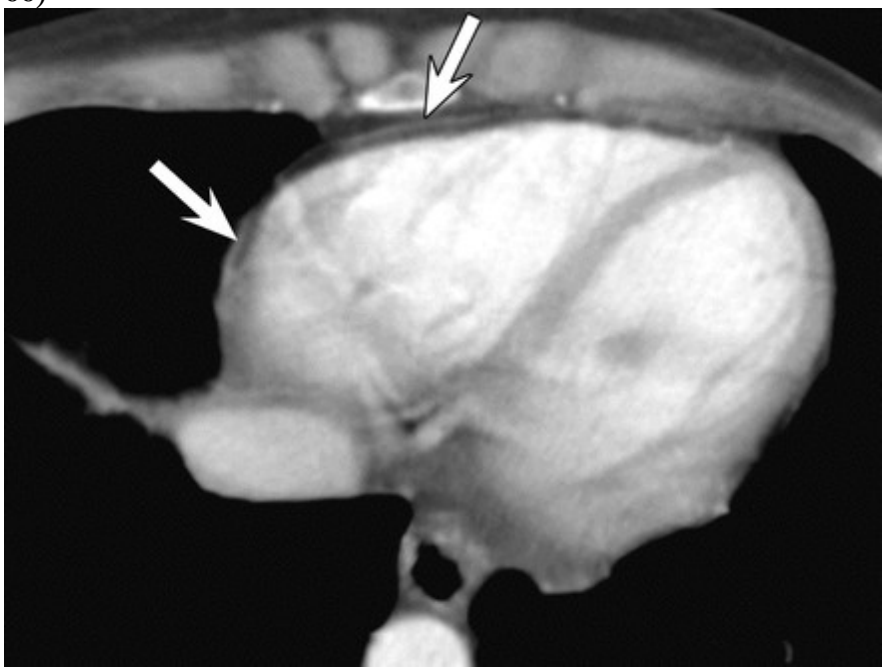


Figure 8 ***Normal pericardium. Axial contrast-enhanced CT scan***

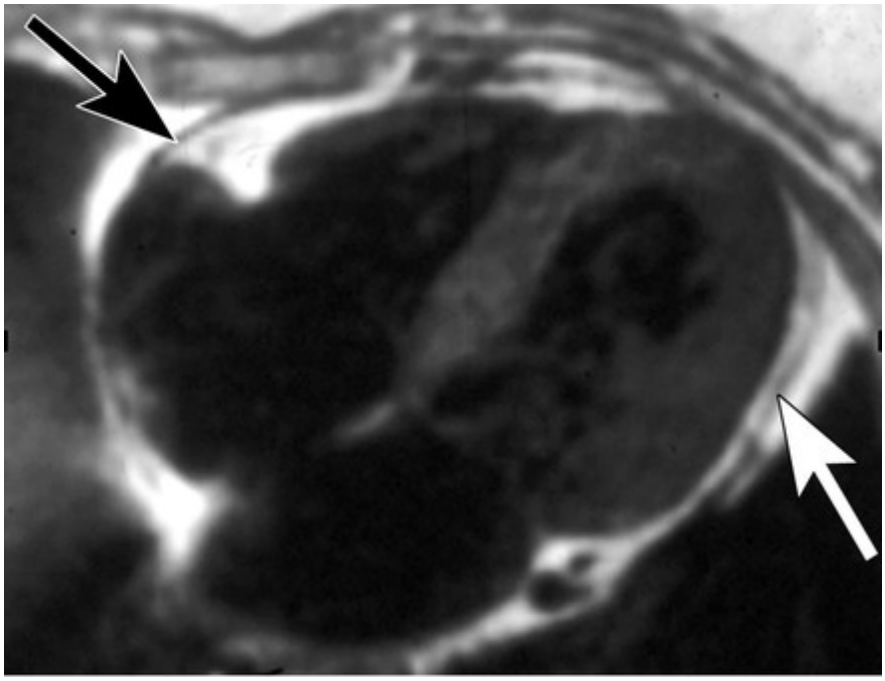


Figure 9 ***Axial ECG-gated spin-echo (SE) MR image (b) show a pericardium with normal thickness***

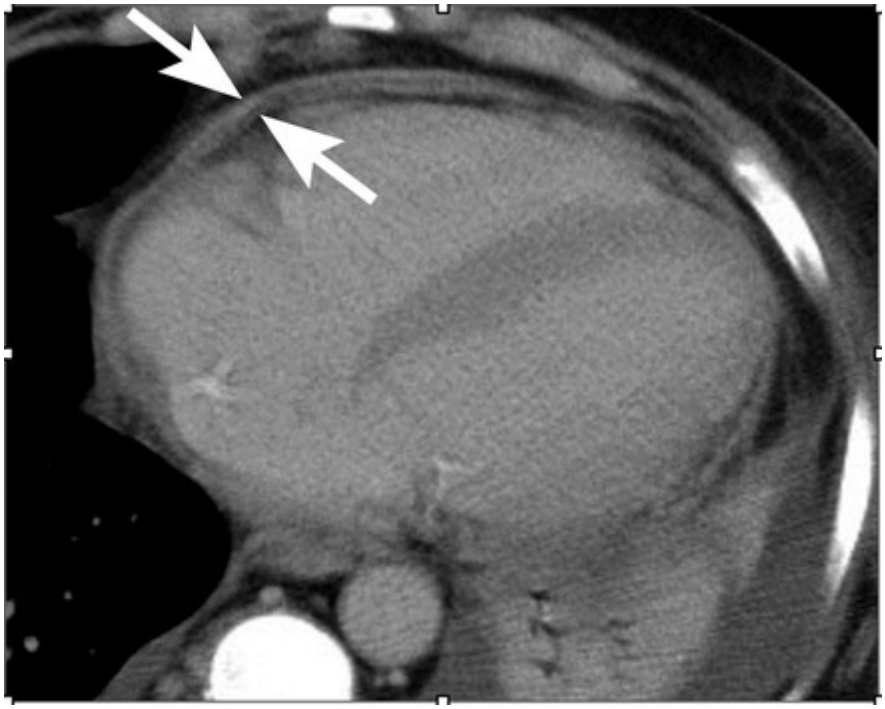


Figure 10 ***Axial contrast-enhanced CT scan shows pericardial thickening (arrows***

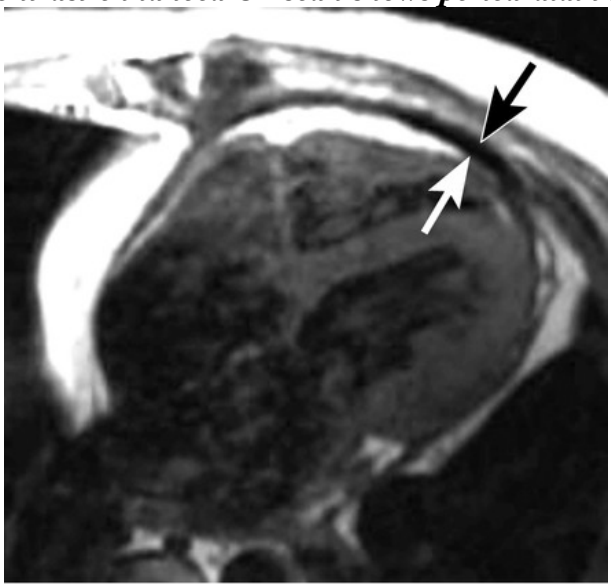


Figure 11. ***Axial ECG-gated T1-weighted SE image shows pericardial thickening (arrows), which is most visible near the right ventricle. The right ventricle has a narrow tubular shape secondary to pericardial constriction***

Figure 12 ***Axial contrast-enhanced CT scan shows dense pericardial calcification (arrows).***

ECHOCARDIOGRAPHIC DIAGNOSIS OF CONSTRICTIVE PERICARDITIS

The two-dimensional and M-mode echocardiographic criteria for CP include abnormal ventricular septal motion (32,33), respiratory variation in ventricular size (34), and presence of a dilated inferior vena cava(34).

The Doppler findings in CP, as described by Hatle et al. (35), are both sensitive and specific (36). They reflect the inspiratory decreases and expiratory increases in left ventricular filling (with reciprocal changes in right ventricular filling) that occur in CP as a result of the relatively fixed cardiac volume. These patients have an expiratory increase in transmitral inflow velocity of 25% or more compared with inspiration. Other characteristic Doppler findings in CP are expiratory decreases in hepatic vein diastolic forward flow and increases in hepatic vein diastolic flow reversals.

TISSUE DOPPLER IMAGING IN CONSTRICTIVE PERICARDITIS

Annulus Paradoxus

Recent observations suggested that mitral annular velocity (E'), as measured by tissue Doppler echocardiography, in patients with systolic dysfunction or primary myocardial disease behaves as a preload-independent index of left ventricular (LV) relaxation (37) and that the ratio of transmitral flow velocity (E) to E' is a predictor of LV filling pressure (38-40) In patients with constrictive pericarditis (CP), however, E' is usually well preserved, despite increased filling pressures (41) possibly due to a greater contribution by the longitudinal movement of the left ventricle for diastolic filling and normal LV relaxation.

The early diastolic velocity of the mitral annulus (E') is reduced in patients with diastolic dysfunction and increased filling pressures. Because transmitral inflow early velocity (E) increases progressively with higher filling pressures, E/E' has been shown to have a

strong positive relationship with pulmonary capillary wedge pressure (PCWP) and left ventricular end-diastolic pressure. However, previous studies have primarily involved patients without a pericardial abnormality. In constrictive pericarditis (CP), E' is not reduced, despite increased filling pressures.
In this classic study by Nishimura et al, Doppler echocardiography was performed to measure early and late diastolic transmitral flow velocities. Tissue Doppler echocardiography was performed to measure E'. PCWP was measured with right heart catheterization. All patients were in sinus rhythm. Mean E and E' were 91±15 cm/s and 11±4 cm/s, respectively. Mean PCWP was 25±6 mm Hg. E' was positively correlated with PCWP (r=0.69, P=0.027). There was a significant inverse correlation between E/E' and PCWP (r=-0.74, P=0.014). Despite high left ventricular filling pressures, E/E' (mean, 9±4) was <15 in all but 1 patient.

Paradoxical to the positive correlation between E/E' and PCWP in patients with myocardial disease, an inverse relationship was found in patients with CP(41,42).

CARDIAC CATHETERIZATION STUDY

Table 8 ***Traditional Hemodynamic Criteria for diagnosing Constrictive Pericarditis*** *(Ref 44-54)*

	Constrictive pericarditis	***Restrictive cardiomyopathy***
End-diastolic pressure equalization	LVEDP−RVEDP≤ 5 mm Hg	LVEDP−RVEDP> 5 mm Hg
Pulmonary artery pressure	PASP< 55 mm Hg	PASP> 55 mm Hg
High RVEDP	RVEDP/RVSP>1/3	RVEDP/RVSP<1/3
Dip-plateau morphology	LV RPW >7mm Hg	LV RPW ≤7mm Hg
Kussmaul's sign	Lack of respiratory variation in mean RAP	Normal respiratory variation in mean RAP

Table 9 ***Dynamic respiratory criteria*** *(Ref 55-58)*

Dynamic Respiratory Criteria

- 5 mm Hg increase in gradient between PCWP and early diastolic LV pressure during expiration compared with inspiration
- Ventricular discordance due to interdependence- inspiratory increase in RVSP and decrease in LVSP; expiratory increase in LVSP and decrease in RVSP.

Table 10 ***Comparison of Traditional and Dynamic Respiratory for Constrictive Pericarditis***

Traditional criteria	***Sensitivity***	***Specificity***	***PPV***	***NPV***
LVEDP−RVEDP≤ 5 mm Hg	60	38	4	57

PASP< 55 mm Hg	93	38	52	89
RVEDP/RVSP>1/3	93	24	47	25
LV RFW > 7mmHg	93	57	61	92
Lack of resp variation in m RAP	93	48	58	92
Dynamic respiratory criteria				
PCWP/LV early filling pressure≥ 5 mm Hg	93	81	78	94
LV/RV Interdependence	100	95	94	100

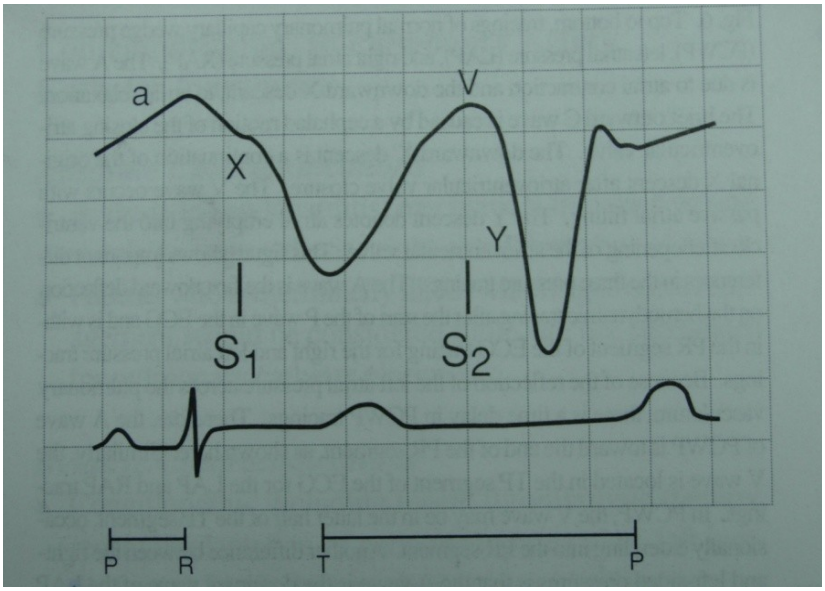


Figure 13 ***RAP tracing in CP***

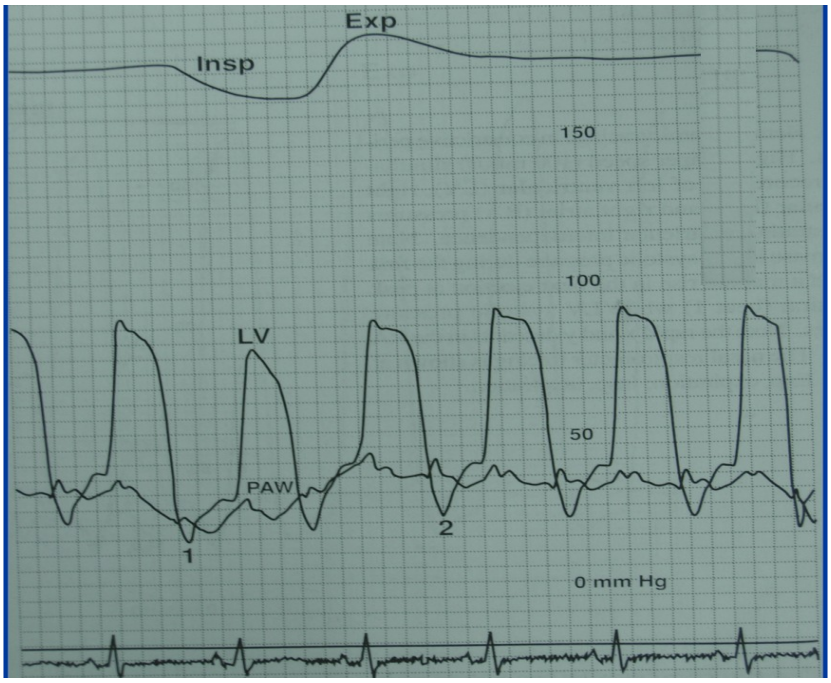


Figure 14 ***Dissociation of Intra-thoracic & Intra-cardiac pressures in CP***

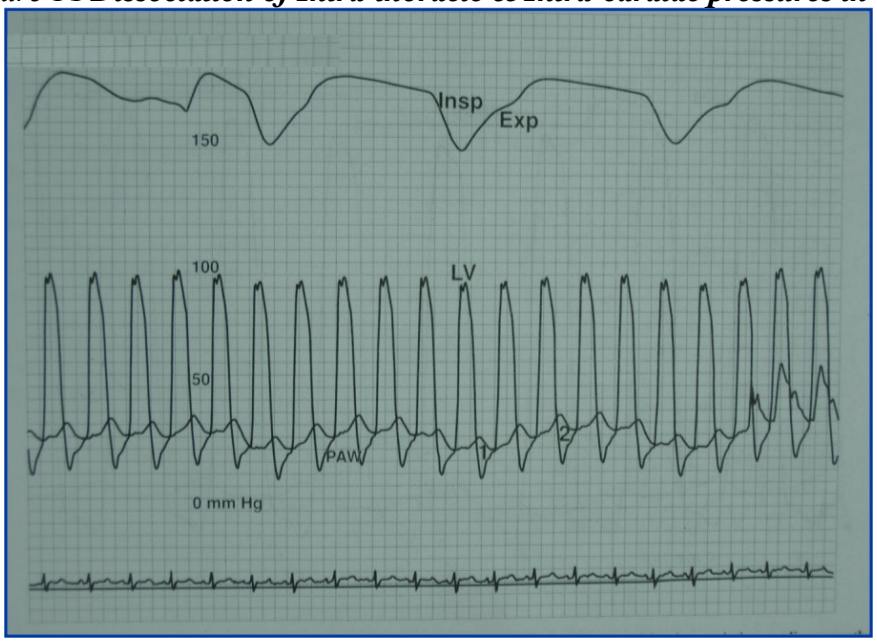


Figure 14 ***Lack of dissociation of Intra-thoracic & Intra-cardiac pressures in RCMP***

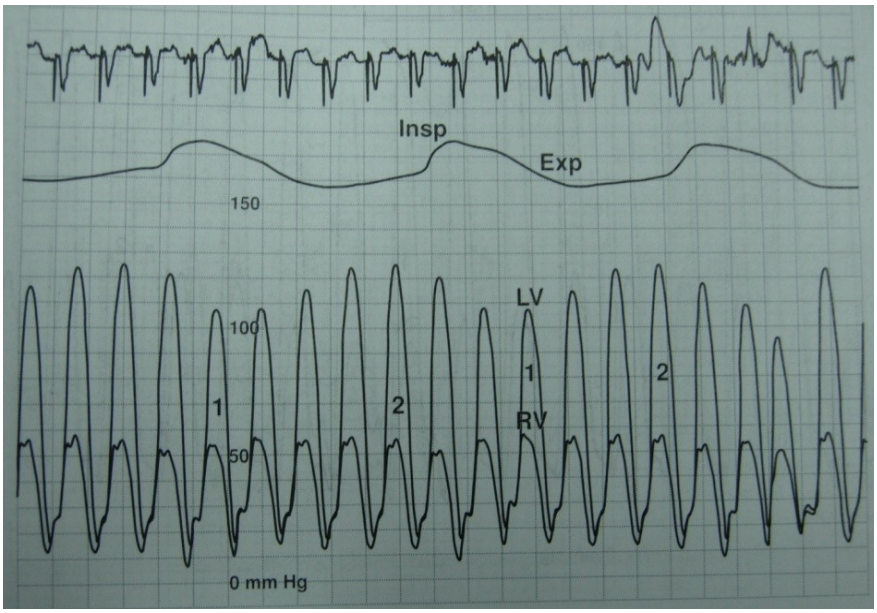


Figure 16 ***.Ventricular pressure discordance in CP***

STUDY DESIGN AND METHODS

Setting	Department of Cardiology, Govt General Hospital, Madras Medical College, Chennai-3
Study design	Single centre, non randomized, observational and prospective study

I. PATIENT POPULATION

The patient population consisted of patients with the clinical diagnosis of constrictive pericarditis who subsequently had surgical confirmation. The research proposal was approved by the institutional review board of ethical committee for clinical research.

These patients all prospectively underwent a rigorously detailed clinical evaluation, biochemical investigation, chest radiography, Computed Tomography (CT) scan of chest and Echocardiography including M-Mode, 2D Echo, Tissue Doppler imaging (TDI) and TransEsophageal Echocardiography (TEE) . The patients subquentely underwent a detailed hemodynamic cardiac catheterization study before confirmation by surgical treatment and pathological confirmation. At operation, all had confirmed constrictive pericarditis by surgical and pathological description.

INCLUSION CRITERIA

All patients with the clinical and Echocardiographic features consistent with the diagnosis of constrictive pericarditis

EXCLUSION CRITERIA

1. Patients with preexisting severe co morbid conditions which may preclude invasive evaluation and surgical treatment.
2. Patients with the Echocardiographic features of thickened pericardium but without clinical and Doppler features of constrictive pericarditis.
3. Patients with associated Rheumatic Heart Disease.

II. STUDY DESIGN

Clinical data

Demographic and clinical profile of the patients were recorded with specific emphasis to signs and symptoms such as

- Dyspnoea and NYHA functional class
- Ascites precox
- Pedal edema
- Fever

- Jugular Venous Pulse and Pressure,Kussmaul's Sign
- Pulsus Paradoxus
- Pericardial Knock
- Hepatomegaly, splenomegaly,
- H/o pericarditis, uremia,malignancy ,previous cardiac surgery
- H/o tuberculosis and anti tubercular treatment, pericardiocentesis, mediastinal irradiation, penetrating and non penetrating chest trauma

ELECTROCARDIOGRAPHIC DATA

12 Lead ECG was analyzed for abnormalities of

- Rhythm
- P wave
- QRS amplitude – low QRS voltage
- Intra ventricular conduction abnormalities
- ST-T changes

CHEST RADIOGRAPHY

Postero Anterior and lateral Chest Radiography was obtained in all patients.
Presence of pericardial calcification was looked for specifically in the lateral view .
Other abnormalities are –

- Presence or absence of cardiomegaly
- SVC distension
- Straightening of right heart border
- Left atrial enlargement
- Pulmonary venous congestion
- Pleural effusion

ECHOCARDIOGRAPHY

The echocardiographic evaluation was done in all patients and the results were analyzed in detail.
Two-dimensional and Doppler echocardiographic examinations were performed in a standard manner with Philips IE33 Echo Machine.
A pulsed-wave Doppler study of mitral inflow velocity was performed. The first cardiac cycles in which filling and ejection occurred in their entirety during a particular respiratory phase (either inspiration or expiration) were analyzed. Three respiratory cycles were analyzed for each patient. For tissue Doppler echocardiography measurements, the specialized pulsed-wave Doppler mode for tissue velocity was used, and the Nyquist limit was adjusted to a range of 15 to 20 cm/s. Gains were minimized to allow for a clear tissue signal with minimal background noise. From the apical 4-chamber view, a 1.5 to2.0-mm sample volume was placed at the septal corner of the mitral annulus to record annular velocities .Similarly tricuspid annular velocity was also recorded. All Doppler velocities were recorded at a sweep speed of 50 or 100 mm/s.

M-MODE

Variables analyzed were

- Pericardial thickness and / or calcification

- Pericardial effusion
- Septal notch
- Abnormal early diastolic flattening of LV posterior wall
- LV dimension and LV EF

2D ECHO DATA

Atrial dilation, LA-LV angle, RV and LV shape, Septal Bounce, Pericardial effusion pericardial thickness and / or calcification, Spontaneous Echo contrast and /or thrombus inside IVC, RA, RAA, Dilated IVC and Hepatic Vein.

PULSED DOPPLER ECHO

Pulsed Doppler of mitral and Tricuspid inflow velocities were obtained by placing a 1–2 mm sample volume between the tips of the mitral and Tricuspid leaflets in the apical four-chamber view. The Doppler beam was aligned parallel to the direction of flow. The following variables were measured both in inspiration and expiration.
Peak early filling velocity (E);
Peak filling velocity at atrial contraction (A velocity);
E/A ratio;
Deceleration time of the peak E velocity, defined as the slope from peak E extrapolated to the baseline value.
Restrictive mitral inflow patterns were defined as E/A ratio >2 with a E-wave deceleration time < 140 ms.

HEPATIC VEIN DOPPLER ECHO

Hepatic vein Doppler study obtained from subcostal view .using 2 to 5 mm pulse wave sample volume placed 1 to 2 cm proximal to the junction with IVC.
The systolic(S) and Diastolic (D) forward flow; systolic flow reversal(SR) and diastolic flow reversal (DR) during inspiration and expiration were assessed .
Also the size and respiratory variation of IVC recorded.

TISSUE DOPPLER IMAGING

TDI of the mitral annulus was obtained from the apical 4-chamber view. A 1.5-mm sample volume was placed sequentially at the lateral and medial mitral annulus. Analysis was performed for the early (E') and late diastolic velocity (A'): peak velocity (E', A'). These variables were analyzed individually, as the average of the medial and lateral annulus.
All Doppler signals were recorded with at100 mm/s. The average of 3 end-expiratory cycles was used. E/E' Ratio was calculated.
Mitral septal annular systolic(S') velocity cut off value of <7.5 cm/s was taken as LV dysfunction.
Similarly septal tricuspid annular systolic(S') velocity was obtained as measure of RV function and cut off value of < 11.5 cm/s was taken as RV dysfunction.
Doppler analysis of pulmonary venous flow was done from left superior pulmonary vein with sample volume of 2 to 5 mm. systolic (S), diastolic (D), S/ D ratio and Atrial reversal (AR) were analyzed.

TRANSESOPHAGEAL ECHOCARDIOGRAPHIC EVALUATION (TEE)

Transesophageal Echocardiography was done in all patients after 2D and Doppler Echo analysis. Pericardial thickness was measured in both mid esophageal 4 chamber view and transgastric view. In addition pericardial calcification, pericardial effusion, strands and AV groove calcification were analyzed.

Pulmonary venous flow (TEE)

The left upper PV was selected for Doppler interrogation, as it lies nearly parallel to the ultrasound beam. Rotating the probe from 0° and 90° in the midesophageal four-chamber view, the left upper PV was visualized just lateral to the LA appendage. Color flow Doppler imaging confirmed its location and the presence of laminar flow. The sample volume was placed centrally, 1 to 2 cm from the vein orifice.
PV systolic flow (S) ,PV diastolic flow (D) and late diastolic retrograde velocity (AR) flow were analyzed both during inspiration and expiration.

Vena cava and Right Atrial Appedage (RAA)

From high esophageal position at 90 to 110 degrees, the SVC and IVC were analyzed along with IAS, RA and RAA for size, spontaneous echo contrast and thrombus. Also the distribution of pericardial constriction was noted.

CARDIAC CATHETERIZATION STUDY

All the patients were screened fluoroscopically for pericardial calcification in PA, Lateral RAO and LAO views.

Cardiac catheterization was performed with the patient in the fasting state. A femoral venous and arterial access site was used in all patients. All patients received 2500 U heparin IV at the start of the procedure.

Right- sided heart pressure waveforms obtained with 6F Cournand catheter advanced retrogradely into the Right ventricle.

Left-sided heart pressure waveforms obtained with 6F pigtail catheter advanced retrogradely into the left ventricle the same calibration procedure was performed using simultaneous and equisensitive transducers.

Right-sided heart catheterization was performed and the catheter was advanced into the pulmonary tree until a pulmonary wedge contour was observed. Confirmation of the wedge position was obtained with an oxygen saturation >95%. For measurement of right-sided heart pressures in all chambers, the right-sided heart catheter was pulled back sequentially into the pulmonary artery, right ventricle, and mid right atrium.

Patients were instructed to inspire deeply during dynamic respiratory measurements. All pressure recordings and tracings were recorded at 25, 50, and 100 mm/s for at least 1

minute at each speed during normal respirations.

Analysis of Pressures: Conventional Criteria

Baseline pressure waveforms were measured simultaneously in the right and left sides of the heart at end expiration, incorporating an average of five consecutive beats. These measurements included the LVSP, RVSP, LVEDP, and RVEDP, mean PCWP, mean pulmonary artery pressure, PASP, MRAP, and mean aortic pressure. Systolic measurements were taken from the peak of the pressure waveform, whereas end-diastolic pressures were measured just before the onset of the ventricular contraction. The height of the RFW was measured from the left ventricular pressure minimum in early diastole to the mid-diastolic pressure plateau.

The previously described "classic" hemodynamic criteria used for the diagnosis of constrictive pericarditis were defined as LVEDP-RVEDP $\leq$5 mm Hg; PASP <55 mm Hg; RVEDP/RVSP >1/3; dip-and-plateau filling, as reflected in the height of the left ventricular RFW; and lack of respiratory variation in the MRAP.

Analysis of Pressures: Dynamic Respiratory Changes

Analysis of the dynamic respiratory changes in hemodynamics was made from the recordings. To assess whether an abnormal dissociation of intrathoracic and intracardiac pressures was present, the PCWP minus the minimum early LVDP gradient was measured during the inspiratory and expiratory phases of respiration. Inspiration was defined as the first ejection beat that followed the first inspiratory diastolic filling period, and the first expiratory ejection beat was the beat after the first expiratory diastolic filling period. The maximal increase and decrease of the PCWP were also measured during the same respiratory cycle.

Evidence for abnormal ventricular interdependence was assessed by analyzing the simultaneous left ventricular and right ventricular waveforms during respiration. The onset of inspiration (beat number one) was defined as the first ejection beat after a decline in early LVDP. Maximum inspiration was defined as the ejection phase of the beat after the diastolic filling phase with the lowest early LVDP. The peak LVSP and RVSP were measured for beat number one as well as the following beats throughout one respiratory cycle.

RESULT ANALYSIS

CLINICAL DATA ANALYSIS

The study consists of 12 patients with clinical, echocardiographic, hemodynamic and surgical diagnosis of constrictive pericarditis .Males predominated in this study by a ratio of 4:1(Table11).

Table 11. Clinical profile of patients with constrictive pericarditis

Name	Age/ sex	NYHAA Class	Duration (months)	Ascites precox	Pedal edema	Fever	Abd. Discom fort	H/o peri carditis	H/o TB, ATT	H/o cardiac Sx	H/o PEF	H/o RT/ Uremia/ malig
1	48/m	III	6	+	+	−	+	−	−	−	−	−
2	40/m	IV	5	+	+	+	+	+	−	−	−	−
3	16/f	II	3	+	−	−	+	−	−	−	−	−
4	17/m	III	18	+	−	−	+	−	−	−	−	−
5	27/f	III	3	−	+	+	+	+	+	−	+	−
6	35/m	IV	2	+	−	+	+	−	−	−	−	−
7	22/m	II	I	+	−	−	+	−	−	−	−	−
8	24/m	IV	12	+	+	+	+	+	+	−	−	−
9	15/f	III	24	+	+	−	+	−	−	−	−	−
10	30/m	II	4	+	−	−	+	+	−	−	−	−
11	29/m	II	5	+	+	+	+	+	+	−	−	−
12	50/m	II	6	+	+	+	+	+	−	−	−	−

Age group of the patients ranged from 15 to 50 years, with a mean age of 28.7 years. The youngest age of the patient was 15 years.

The mean duration of symptoms was 7.4 months (range: 1 to 24 months, median: 5

months).

The most common presenting feature was dyspnoea and abdominal distension which were present invariably in all patients. Pedal edema was present in 7 patients (58.3%). Ascites precox was the next most common (91.6%) feature. Also abdominal distention and loss of appetite was present in all patients. History of fever with chest pain suggestive of pericarditis was present in half of the patients.

Two patients gave history of anti TB treatment for pericardial effusion at the onset of illness. None of the patients of the study group had prior cardiac surgery, mediastinal irradiation, uremia or malignancy as a cause for constrictive pericarditis.

On physical examination, (Table 12) 11 patients (91.6%) had elevated jugular venous pressure range 10 to 15 cm of water. Jugular venous wave form showed prominent y wave (y>x) in 9 patients(75%).The x and y waves were equally prominent in 2 patients. In one patients only the prominent y but not x wave could be observed.

Table 12 ***.Clinical Signs Profile in patients with constrlictive pericarditis***

Name	JVP cm	JV wave	Kussmaul sign	Pulsus paradoxus	Ascites	Edema	Jaundice	Peri knock	murmurs	Liver	Spleen
1	10	X<Y	+	−	+	+	−	+	−	+	+
2	15	X<Y	−	−	+	+	−	+	−	+	+
3	8	X<Y	+	−	+	−	−	+	−	+	−
4	15	X<Y	+	−	+	−	−	+	−	+	−
5	13	X=Y	+	+	+	+	−	−	−	+	−
6	12	X=Y	+	+	+	−	+	+	+	+	+
7	10	Y	+	−	+	−	−	−	+	+	−
8	15	X<Y	+	−	+	+	+	+	−	+	+
9	12	X<Y	+	−	+	+	−	+	−	+	−
10	14	X<Y	+	+	+	−	−	−	−	+	+
11	11	X<Y	+	−	+	+	−	+	+	+	−
12	12	X<Y	+	−	+	+	−	+	−	+	−

The absence of inspiratory decrease or inspiratory increase (in JVP) of Kussmaul‘s sign was present in 10 patients (84%).
Pulsus Paradoxus present in one fourth of the patients.
Tense ascites was present invariably in all patients. Pedal edema was absent in 5 patients (41%).
The early diastolic pericardial knock was present in 9 patients (75%) and systolic murmur of mitral regurgitation was present in 3 patients (25%). There was no audible murmur of tricuspid regurgitation or pericardial rub
Hepatomegaly was invariable but spleen was palpable in one third of the patients.

ANALYSIS OF INVESTIGATIVE DATA

Electrocardiography (Table- 13)

No patient was in atrial fibrillation; all patients were in sinus rhythm. Sinus tachycardia (HR range 100 to130 pbm) was present in 10 patients (83.3%); in 2 patients the heart rate was within normal range.

Table 13 ***ECG findings in patients with constrictive pericarditis***

Name	*Rate/bpm*	*Rhythm*	*Notched P wave*	*Low QRS voltage*	*IVCD*	*ST depression*	*T wave inversion*
1	100	Sinus	+	−	−	V4-V6	L II,III,aVF V4-V6

2	112	Sinus	+	+	−	V2-V6	L I,II,III,aVF V4-V6
3	134	Sinus	−	−	−	V2-V6	L II,III,aVF V2-V6
4	120	Sinus	−	+	−	V4-V6	L II,III,aVF V2-V6
5	100	Sinus	−	+	−	V1-V3	V1-V3
6	130	Sinus	−	+	−	V2-V6	L II,III,aVF V3-V6
7	106	Sinus	+	−	−	V5-V6	L II,III,aVF V4-V6
8	124	Sinus	+	+	−	V2-V6	L II,III,aVF V2-V6
9	120	Sinus	+	+	−	V3-V6	Flat LI,II,V4-V6 ↓V2 –V3
10	110	Sinus	−	−	−	V2-V4	V2-V6
11	75	Sinus	−	−	−	L II,III,aVF V2-V5	L II,III,aVF V2-V5
12	84	Sinus	+	+	−	V5-V6	L I,II,III,aVF V5-V6

Abnormal notched P wave was present in 50% of the patients. And low voltage QRS complexes in 7 patients (58.3%). There was no significant conduction abnormalities observed.

The ECG was abnormal in all the patients. ST segment in precordial leads and inverted T wave abnormality were present in all the patients. Flat T wave was present in one patient along with other changes .No patient had normal ECG.

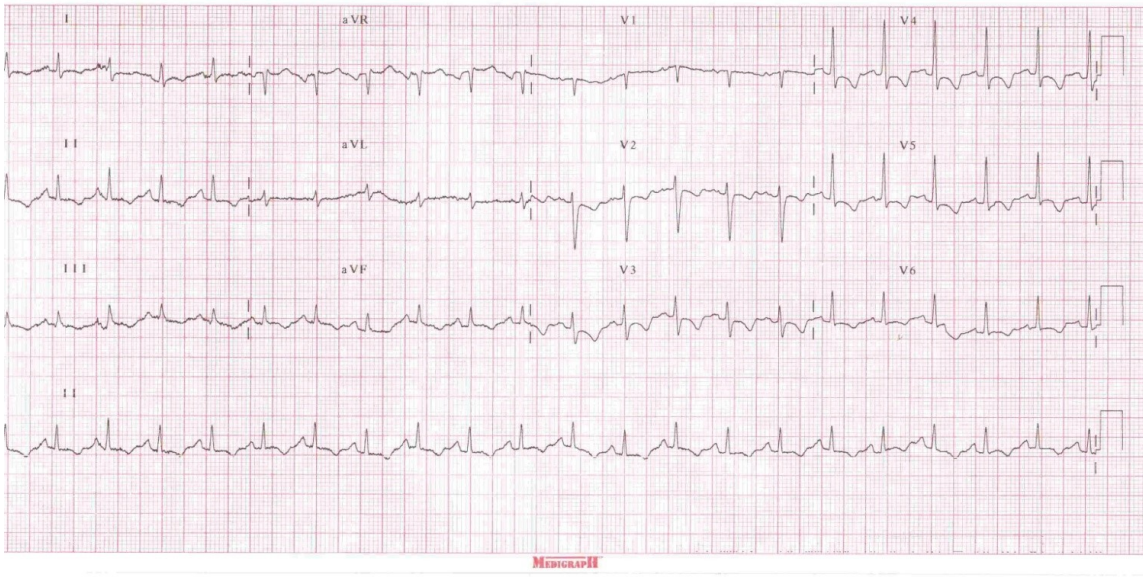


Figure 17 ECG of a patient with constrictive pericarditis showing non specific ST-T changes

Chest Radiography (Table-14)

Heart size was normal in one third of the patients (CTR ≤0.50). In remaining two third of the patients mild to moderate cardiomegaly was present (CTR range: 0.55 to 0.60).

Table 14 ***.Chest Radiography findings in patients with constrictive pericarditis***

Name	***CTR%***	***Straight RHB***	***Peri calcific***	***LAE***	***Pleural eff***	***Pulm Venous cong***	***SVC***
1	0.53	+	Diaph surface	−	left	−	−
2	0.58	−	−	−	right	+	+
3	0.50	−	−	−	right	−	+
4	0.50	−	Diaph surface	−	right	+	+
5	0.55	+	−	−	left	+	+
6	0.60	−	−	−	right>left	+	+
7	0.60	−	−	+	−	+	+
8	0.55	+	−	−	−	+	+
9	0.55	+	−	+	right	+	+
10	0.55	−	−	−	left	−	+
11	0.50	−	−	−	left	−	+
12	0.55	+	−	−	left	+	−

Straightening of the right heart border was present in 5 patients (41.6%) . Left atrial enlargement was present in two patients (16.6%). Pulmonary venous congestion was present in 8 patients (66.6%).Severity of pulmonary venous congestion was Grade I to II.Frank pulmonary edema was not seen. Distention of SVC was present in 10 patients(83%).Pericardial calcification was present in two patients, (16.6%) appears as thick shaggy calcification of pericardium over Right ventricle, diaphragmatic surface and apex of LV. Calcification was best seen in lateral view.

Pleural effusion was present in 10 patients (83%).It was bilateral in one patient (8.3%) with right more than left. Remaining patients had unilateral pleural effusion with left sided effusion in 5 patients (41.6%) and right sided pleural effusion in 4 patients (33.3%).

Figure 18 A

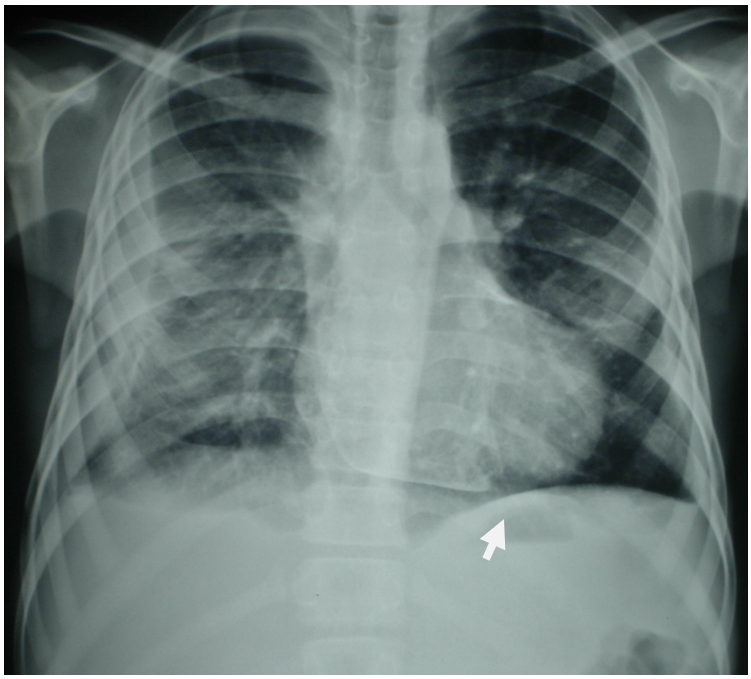
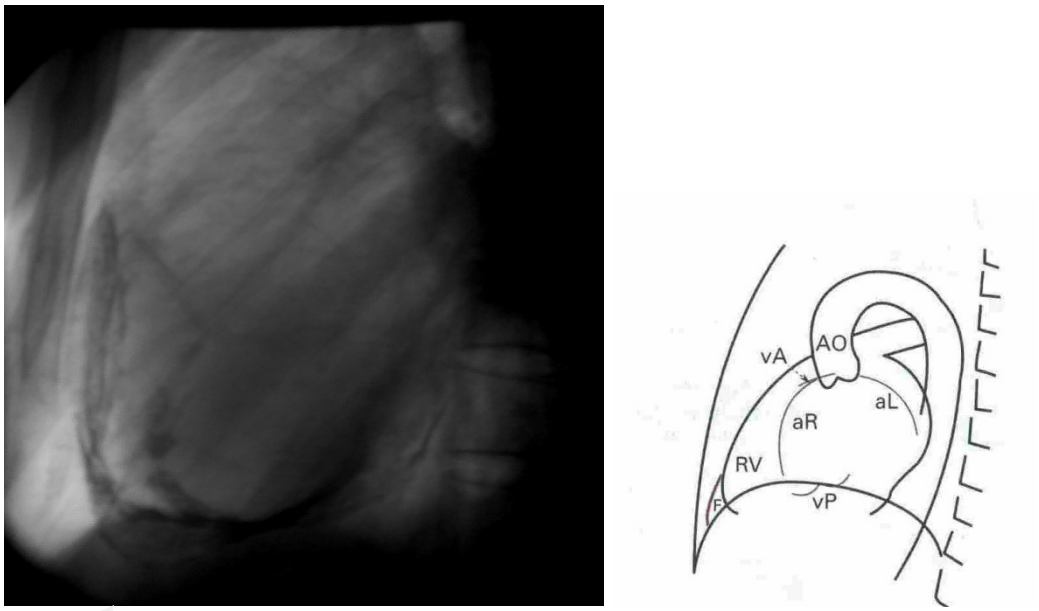


Figure 18B

Figure 18 A,B Chest Radiography PA and Lateral views show pericardial calcification



Figur. 19 Pericardial calcification seen by fluoroscopy (Lateral view)

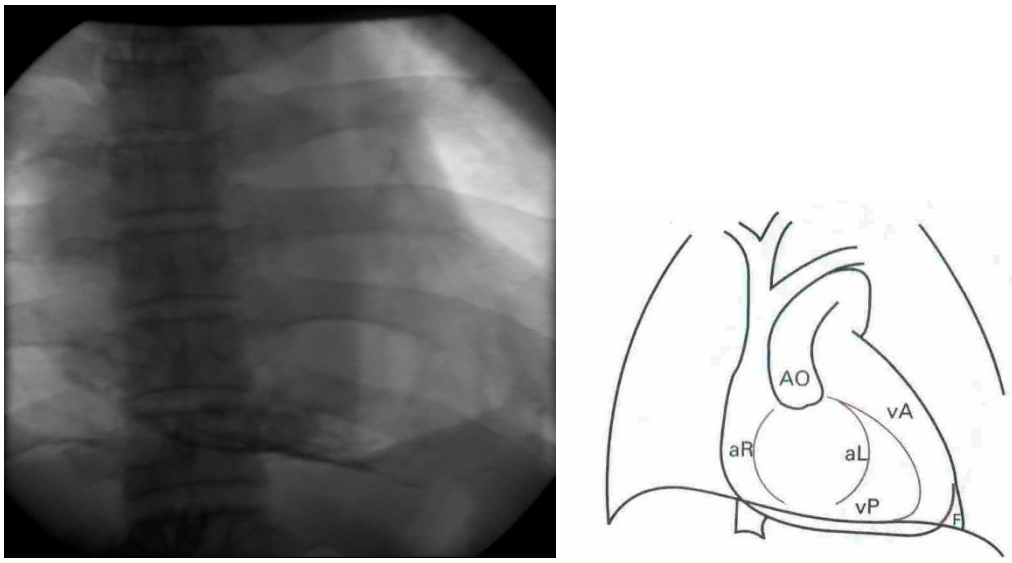


Figure 20 Pericardial calcification seen by fluoroscopy (PA view)

Computed Tomographic (CT) scan (Table-15)

CT scan of the chest was obtained in all patients with and without intravenous contrast agents. Pericardial effusion was present in 7 cases (58.3%).Effusion was mild in 3 patients (25%) and minimal in 2 patients (16.6%). Two patients had moderate pericardial effusion with thick strands in the pericardial cavity. Pericardium was thickened more than 4 mm in 11 patients (91.7%). The thickness ranged from 4 mm to 35 mm .In one patient pericardial thickness was about 4 mm. The median pericardial thickness was 15 mm. The pattern of distribution of pericardial thickening was as follows: global in 5 patients, one patient had predominant thickening over LV.Pericardial thickening over RA/RV>LV was present in 3 cases. Remaining three patients the maximal thickness was over the RA.

Maximal thickness around RA was 33 mm.

Table 15 ***.CT scan findings in patients with constrictive pericarditis***

Name	*PEF*	*Peri Thickening (mm)*	*Distribution of thickening*	*Peri calcific*	*Tube like chambers*	*IVC dilation*	*Pleural effusion*
1	−	10	RA/RV>LV	+	RV	+	L
2	+	12	RA/RV>LV	+	RV	+	R>L
3	+	20	RA>LV>RV	−	−	+	R
4	+	16	RA/RV>LV	−	−	−	R
5	++	17	RA>RV>LV	−	−	−	R>L
6	+	18	Global	−	−	+	R>L
7	+	10	Global	−	−	+	−
8	−	15	Global	−	−	+	R
9	−	10	Global	+	−	+	R>L
10	++	25	Global	−	−	+	L>R
11	+	04	LV	−	−	−	R>L
12	−	14	RA/RV>LV	+	RV	+	L

Pericardial calcification was present in one third of the patients. Calcification was over RV and Right AV groove in 3 patients (25%)and extensive involving RV, LV and Right AV groove in 1 patient.

Deformed chambers appearing as tube-like structure was present in 5 patients (41.6%)- deformed LV 2 patients ; deformed RV 3 patients.IVC dilatation was present in 7 cases (58.3%). Pleural effusion was present absent in one patient; bilateral in 6 patients (50%); unilateral (R or L) in 5 patients (41.6%) .RA or RV thrombus was not seen in any of the patients.

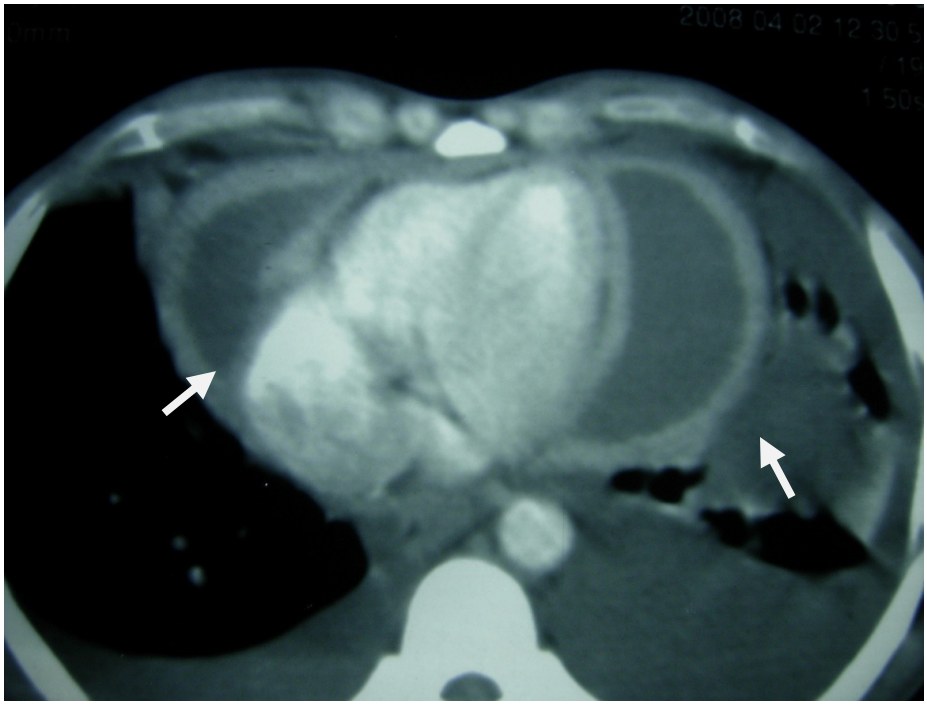


Figure 21 Contrast enhanced CT chest showing markedly thickened pericardium without calcification in a patient with constrictive pericarditis (Global form)

Figure 22 Constrictive pericarditis- Contrast enhanced CT chest showing markedly thickened pericardium especially around the Right Atrium.

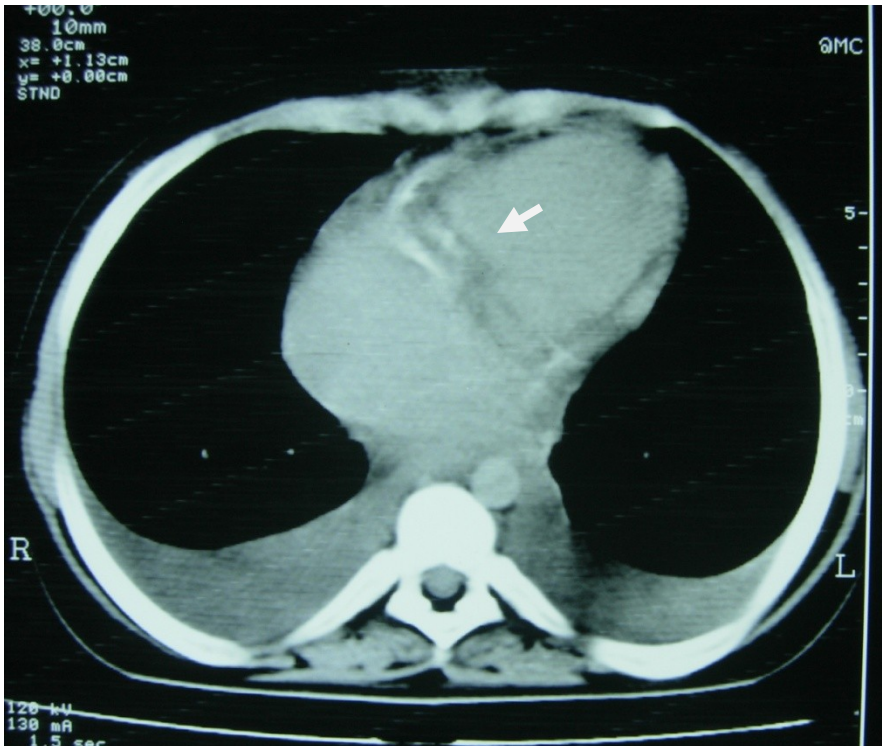


Figure 23 A Contrast enhanced CT chest showing thickened pericardium with Rt AV groove calcification in a patient with constrictive pericarditis. Bilateral pleural effusion is present.

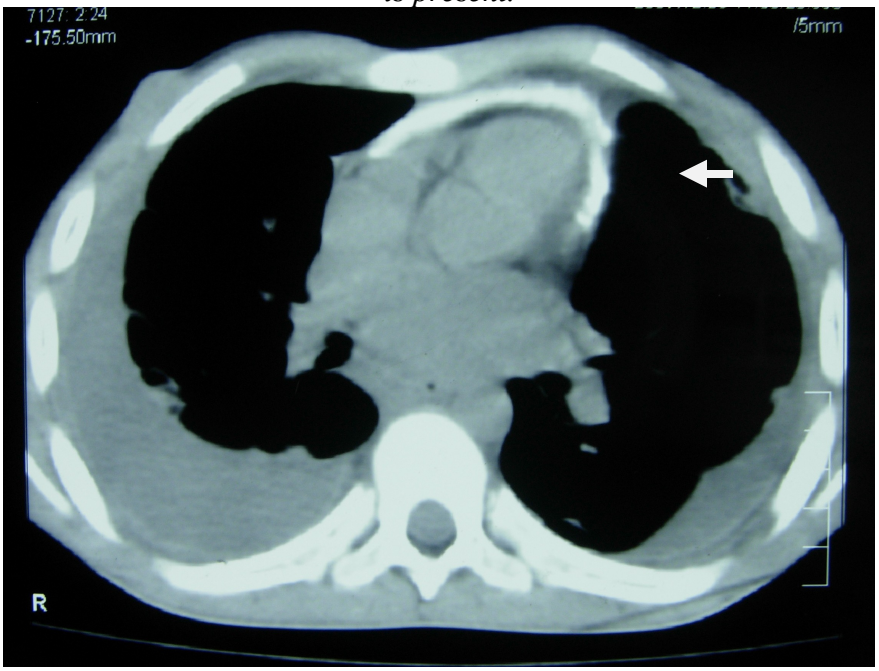


Figure 23 B Contrast enhanced CT chest showing thickened pericardium with calcification around RV,LV and LV apex.

ECHOCARDIOGRAPHIC DATA ANALYSIS

M-Mode Echocardiography (Table-16)

The M-Mode features of classic constrictive pericarditis such as thickened pericardium, early diastolic flattening of LVPW and septal notch was present in all cases .The presence of calcification could be identified by the M mode echo in 2 patients. LV systolic function was normal in 7 patients. There was mild LV dysfunction (EF range 44% to 53%) in 5 patients (41.6%).there was no LV dilatation. Left atrial enlargement was present in one patient. The mean LVEDD was 3.9 cm (range: 3.0 to 4.6 cm).

Table 16 **. M-Mode**

Name	Peri Thickness (mm)	Peri calcific	PEF(mm)	LV Dimension(cm) LVEDD/LVESD/EF%	LVPW flat	Septal notch
1	8.1	+	+	4.5/3.3/53%	+	+
2	14	−	+	4.6/3.4/50%	+	+
3	Ant 11 Post 15	−	Mild	3.7/2.5/62%	+	+
4	6.6	−	−	3.2/2.4/52%	+	+
5	Post 19	−	Ant 10 Post 15	3.4/1.9/65%	+	+
6	11.4	−	++	4.2/3.1/49%	+	+
7	Ant 3.5 Post 12.9	−	+	4.4/3.0/60%	+	+
8	Ant 9.3 Post 23.1	−	−	4.1/2.8/62%	+	+
9	16	−	+	3.0/1.9/69%	+	+
10	39	−	+	3.6/2.8/44%	+	+
11	12	−	−	3.9/2.6/64%	+	+
12	15.7	+	Ant 3 Post 6	3.8/2.6/58%	+	+

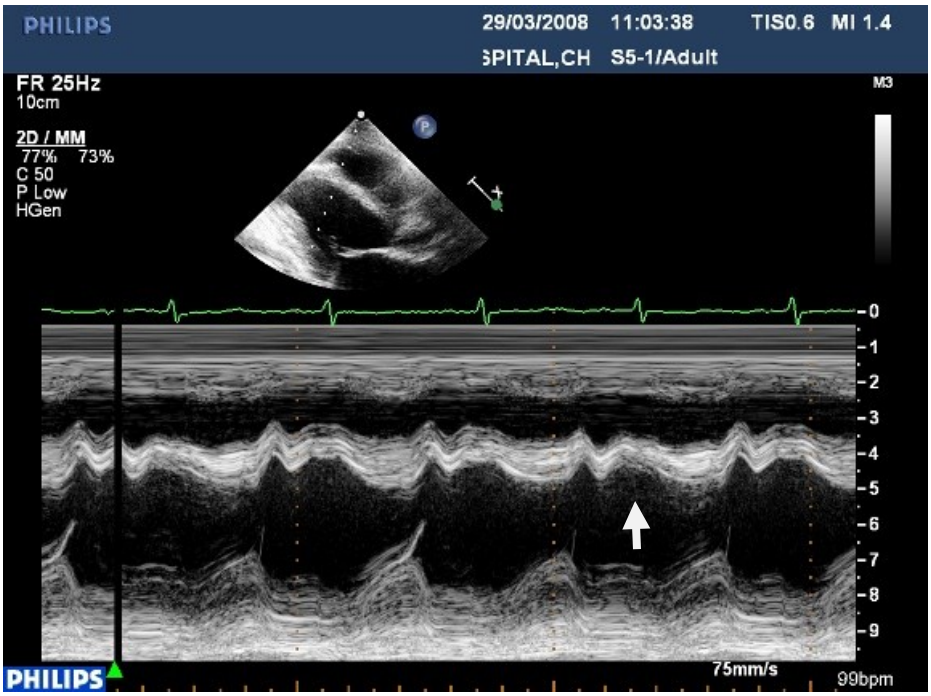


Figure 24 ***M-Mode echo showing prominent early diastolic septal notch (Spanish notch), abrupt flattening of LVPW and thickened pericardium (arrowhead)***

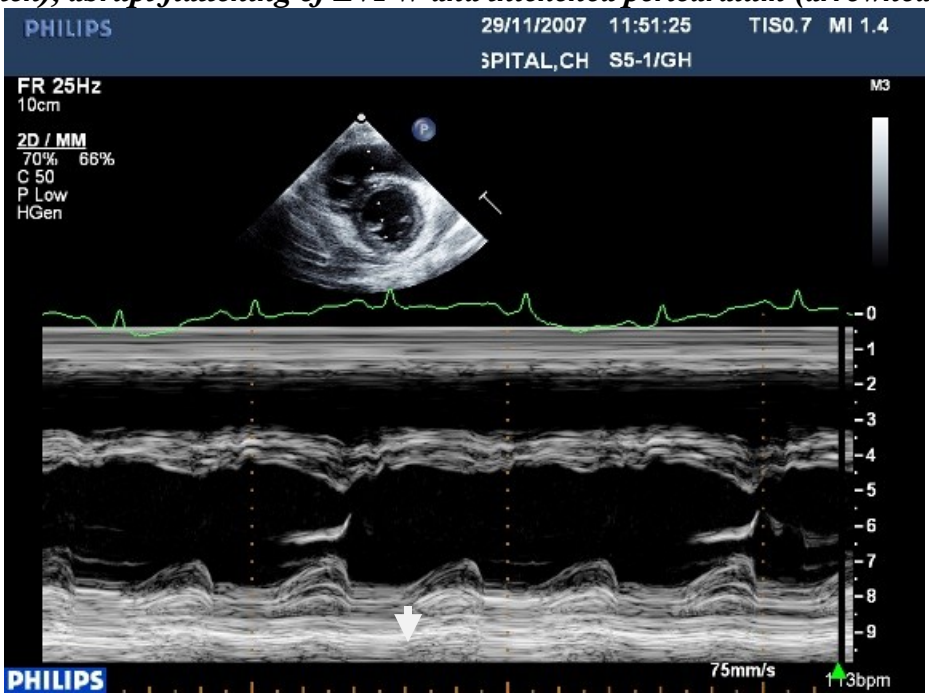


Figure 25 ***M-Mode echo showing abrupt flattening of LVPW and thickened pericardium (arrow)***

2D Echocardiography (Table- 2,17)

The echocardiographic signs such as septal bounce and dilated IVC were present in all the patients. Spontaneous echo contrast inside the cardiac chambers – RA and / or RV was present in 6 patients. (50%).Spontaneous echo contrast inside the dilated IVC was

seen in 9 patients (75%).

Table 17. 2D Echo

Name	*Atrial dil*	*LA-LV angle*	*Tube Like chambers*	*PEF*	*Peri calcific*	*Septal bounce*	*IVC dil*	*Thrombus*	*SEC*
1	−	obtuse	RV	−	+	+	+	−	IVC/ RA
2	−	obtuse	−	−	−	+	+	−	−
3	−	obtuse	RV	+	−	+	+		
4	+	obtuse	LV	−	−	+	+	RA/RV	IVC/RA/RV
5	−	obtuse	−	+		+	+		
6	−	obtuse	−	++	−	+	+	IVC/ RA/RAA	IVC/RA/RV
7	−	obtuse	−	+	−	+	+	−	−
8	−	obtuse	RV	Min RA/RV	−	+	+	−	
9	−	obtuse	−	RA/RV/LV	R AV groove	+	+	RA	IVC/ RA
10	−	obtuse	−	++	−	+	+	−	IVC/RA
11	−	obtuse	−	−		+	+	−	IVC
12	−	obtuse	LV	+	+	+	+	−	RA

Mean size of IVC was 2.08 cm during inspiration (range; 1.55 to 2.53) and 1.8 cm during expiration (range; 1.43 to 2.26 cm). All patients had <50% respiratory variation of IVC and in 3 patients (25%) IVC showed absence of respiratory variation.

LA- LV angle was abnormal in one patient.

Pericardial calcification was evident in 3 patients.

Thickened pericardium was seen in 11 patients. Deformed ventricular morphology – tube like chambers was present in 5 patients (41.3%).with tube like RV in 3 patients and LV in 2 patients.

Pericardial effusion was present in 8 patients (66.6%).

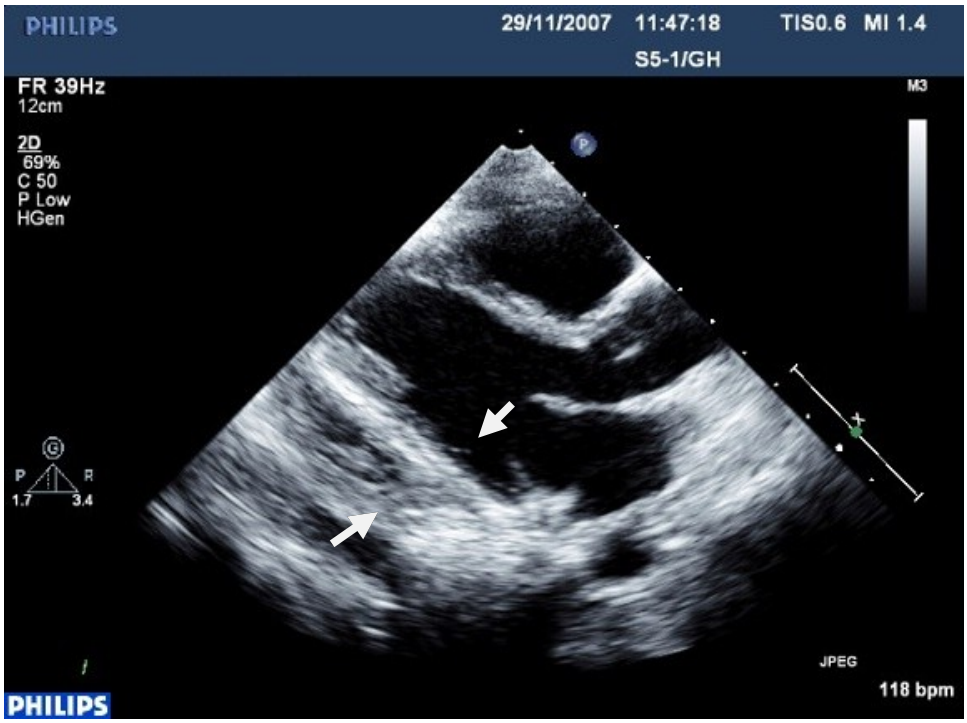


Figure 26 PLAX view showing minimal pericardial effusion with thickening

Doppler Echocardiography (Table-18)

Mitral early diastolic peak velocity (E) analysis

The mean Mitral early diastolic peak velocity (E) analysis showed mean value of 52.2 cm/s (range: 32.1 to 78 .0) during inspiration and 70.7 cm /s (range: 38.5 to 100.1) during expiration.

Table 18. ***Doppler Echo***

	Mitral						***Tricuspid***		
	E Velocity (cm/s)			***A Velocity***			***E Velocity (cm/s)***		
Name	***Ins***	***Exp***	***Resp varia (%)***	***Ins***	***Exp***	***E/A***	***Ins***	***Exp***	***Resp varia (%)***
1	32.1	38.5	20%	25.8	29.5	1.30	36.8	23.6	55.9%
2	39.6	62.5	57.8%	28.6	32.1	1.12	58.6	39.5	48.3%
3	37.7	54.5	44.5%	30.9	33.9	1.6	39.1	34.6	13%
4	74.9	100.1	33.6%	41.1	53	1.88	86.6	63.7	35.9%
5	66.2	82.7	25%	42.9	43.3	1.9	52.0	32.0	62.5%
6	46.3	65.1	40.6%	20.3	23.2	2.8	57.7	37.3	54.5%
7	78.0	97.5	25%	62	72	1.35	81.4	57.0	42.8%
8	39.8	58.6	47.2%	30	38.6	1.51	69.4	48.6	42.8%
9	51.9	80.5	55.1%	24.4	26.8	3.0	58.9	43.8	34.4%
10	48	64.5	34.3%	44.6	46.5	1.38	47.2	31.5	49.8%
11	76	90.8	19.4%	37	42	2.1	48.	32.	48%

						6	4	7	
12	36.2	53.6	48%	35.5	49.4	1.08	65.7	54.9	19.6%

Respiratory variation in the Mitral early diastolic peak velocity (E) of > 25% increase during expiration compared to inspiration was present in 10 patients (88.3%).The variation ranged from 25% to 57.8% .

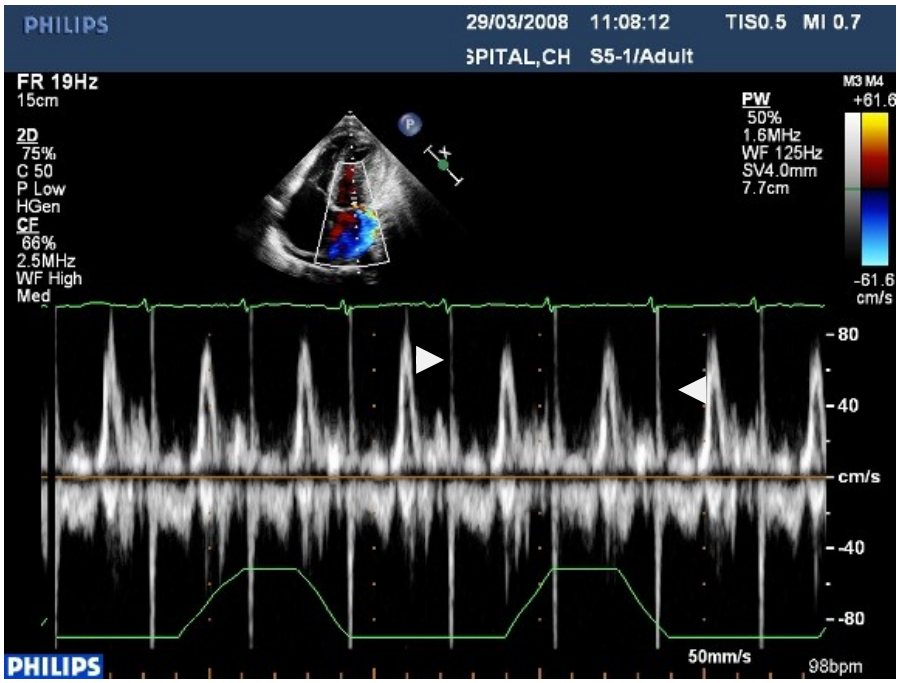
The ratio between Mitral early diastolic peak velocity (E) and Mitral late diastolic peak velocity (A) was > 2.0 was present in 3 patients (25%).

Tricuspid early diastolic peak velocity (E) analysis

The mean value of Tricuspid early diastolic peak velocity (E) was 58.4cm/s (range:30.8 to 86.6) during inspiration and 41.6 cm/s (range 23.6 to 63.7) during expiration.

Respiratory variation in the Tricuspid early diastolic peak velocity (E) of >40% increase during inspiration compared to expiration was present in 8 patients (66.6%). The variation ranged from 13% to 62.5%.

A



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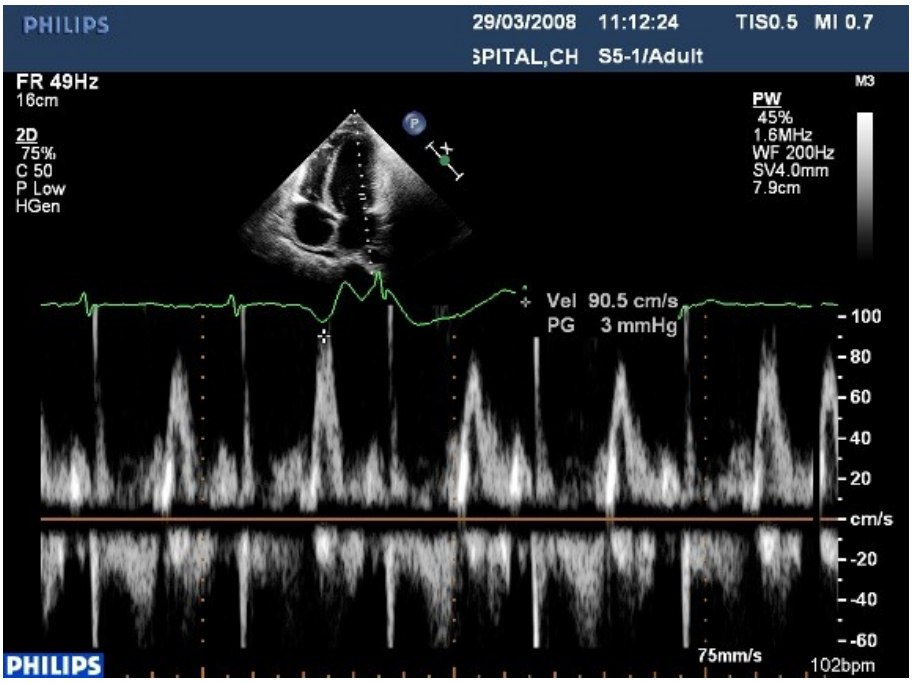

Figure 27 A,B Respiratory variation in the Mitral early diastolic peak velocity (E) increase during expiration compared to inspiration.

Figure 28.Respiratory variation in the Tricuspid early diastolic peak velocity (E) increase during inspiration compared to expiration

Pulmonary Venous Doppler analysis (Table-19)

Pulmonary venous systolic flow (S) velocity

The Pulmonary venous systolic flow (S) velocity was lower during inspiration (mean 27.5 cm/s, range: 17.7 to 40.7) than during expiration (mean 32.6 cm/s, range: 19.6 to 48.7).

Pulmonary venous diastolic flow (D) velocity

The Pulmonary venous diastolic flow (D) velocity ranged from 29.7 to 42.5cm/s (mean 35.9) during inspiration and 38.8 to 68.2 cm/s (mean 47.3) during expiration.

Ratio(S/D) between Pulmonary venous systolic flow (S) velocity and Pulmonary venous diastolic flow (D) velocity

was 0.75 (range:0.50 to1.05) during inspiration and 0.75 (range:0.48 to 0.90) during expiration .

The S/D ratio was <1.0 in all patients. It was >0.65 in 10 patients (83.3%)and one patient had value of <0.50 .

The respiratory variation in Pulmonary venous diastolic flow (D) velocity of > 40% increase during expiration was present two patients (range :10.4% to 76.2%)

Table 19. ***Pulmonary Venous Doppler (By TEE)***

Name	***Systolic(S) Cm/s***		***Diastolic(D) Cm/s***		***S/D ratio***		***(D) Resp Variation %***	***Atrial Reversal (AR) Cm/s***
	Ins	***Exp***	***Ins***	***Exp***	***Ins***	***Exp***		
1	22.0	26.8	29.7	38.5	0.74	0.69	29.6%	14.5
2	40.7	48.7	38.7	68.2	1.05	0.71	76.2%	24.3
3	23.3	28.5	31.5	38.8	0.73	0.73	23.1%	12.9
4	32.4	41.5	37.4	46.8	0.86	0.88	25.1%	46.2
5	17.7	19.6	32	40.5	0.55	0.48	26%	34.2
6	29.6	38.6	38.4	49.2	0.77	0.78	28.1%	32.5
7	24.8	30.6	30.8	41.5	0.80	0.73	34%	30.8
8	39.2	46.8	42.5	51.6	0.92	0.90	21.4%	28.5
9	23.1	32.5	31.3	40.7	0.69	0.79	30%	13.0
10	20	29.1	33.0	45.7	0.60	0.63	38.4%	23.1
11	25.4	46.4	41.3	57.6	0.61	0.80	39.4%	17.6
12	32.2	42.0	43.9	48.5	0.73	0.86	10.4%	14.1

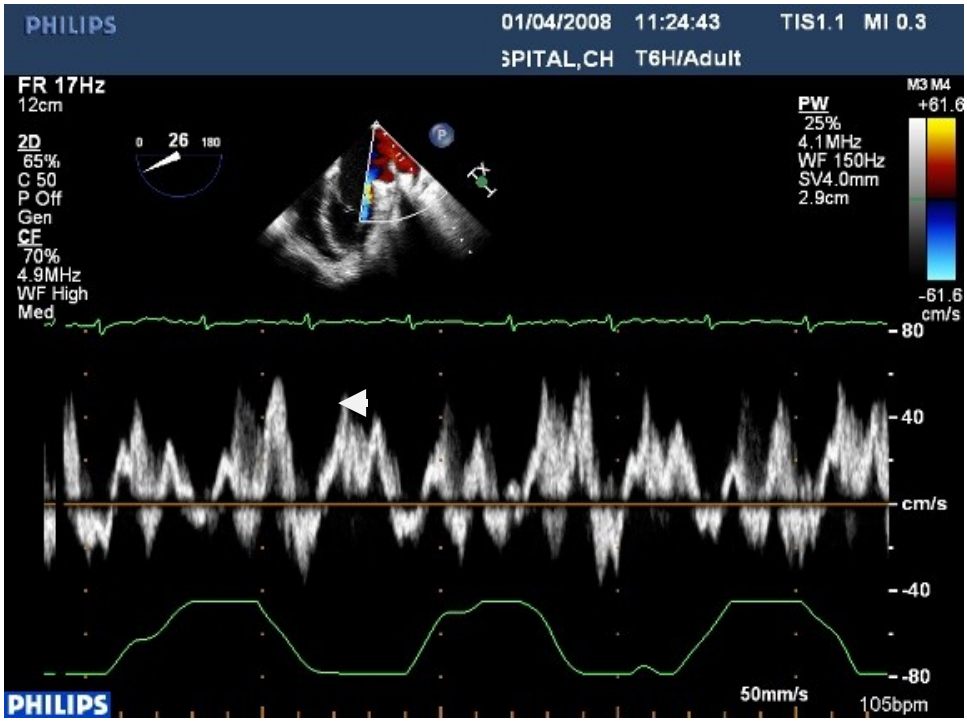

Figure 29 Pulmonary venous doppler-expiratory increase in diastolic (D) velocity

Hepatic Venous Doppler Analysis (Table-20)

Hepatic Venous systolic(S) flow analysis showed an increased flow velocity during inspiration than during expiration Inspiration: mean S 54.8 cm/s (range: 19.1 to 38.6).
Hepatic Venous Systolic flow reversal (SR) showed expiratory exaggeration with mean value of 15.9 cm/s (range:8.2 to 29.3) during inspiration and mean value of 19.2 cm/s (range:9.5 to34.2) during expiration.
Hepatic Venous diastolic (D) flow analysis showed mean value of 22cm/s (range:10.6 to 40.1).
Analysis of Hepatic Venous diastolic flow reversal (DR) analysis during inspiration and expiration revealed an exaggerated expiratory increase of diastolic flow reversal (9.7% increase during expiration).
Diastolic flow reversal (DR) during inspiration – mean 21.4 cm/s (range: 13.3 to 32.8)
.Diastolic flow reversal (DR) during expiration – mean 31.1 cm/s (range: 21.6 to 55.1).

Table 20 Hepatic Vein Doppler

Name	*Systolic(S) cm/s*	*Systolic Reversal(SR) cm/s*		*Diastolic(D) cm/s*	*Diastolic Reversal(DR) cm/s*	
		Insp↑	*Exp↑*		*Insp↑*	*Exp↑*
1	25.7	8.2	9.5	17	18	23.7
2	22.3	11.6	16.8	15.8	19.6	27.5
3	21.9	13.4	15.4	15.1	18.4	25.8
4	36	13.6	24.3	40.1	32.4	55.1
5	26.2	18.5	27.9	14.5	13.3	26.4
6	19.1	15.7	19.5	28.4	32.8	36.8
7	19.7	14.4	26.1	18.6	24.5	34.2
8	38.6	29.3	34.2	28.7	25	30.7
9	24.9	11.2	10.9	10.6	17.2	35.3
10	28.9	18.8	11.8	18.3	16.5	26.2
11	22.2	17.6	18	19.1	15.6	21.6
12	21.7	18	15.5	38.3	23.9	31.9

Table 21 IVC: M- Mode& 2D-Echo

Name	*IVC size (cm)*		*Respiratory Variation*			*Estimated RAP (mm Hg)*	*SEC*
	Ins	*Exp*	*>50%*	*<50%*	*Absent*		
1	2.53	2.12	−	+	−	15-20	+
2	2.45	2.26	−	+	−	15-20	+
3	1.70	1.70	−	−	+	10-15	−
4	1.60	1.60	−	−	+	10-15	+
5	1.55	1.43	−	+	−	10-15	−
6	1.93	1.90	−	−	+	10-15	+
7	1.78	1.44	−	+	−	10-15	−
8	1.84	1.76	–	+	−	10-15	+
9	2.48	1.35	−	+	−	15-20	+
10	2.5	2.3	−	+	−	15-20	+
11	2.19	1.8	−	+	−	10-15	+
12	2.21	1.92	−	+	−	10-15	+

Figure 30 Hepatic Vein Doppler – exaggerated diastolic reversal (DR)during expiration(double arrow head)

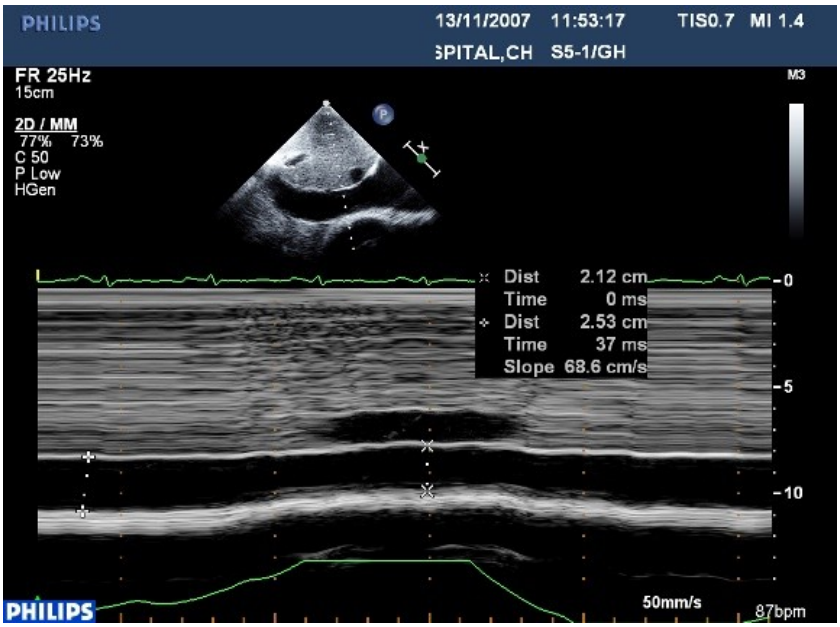

Figure 31 Dilated IVC with <50% inspiratory collapse

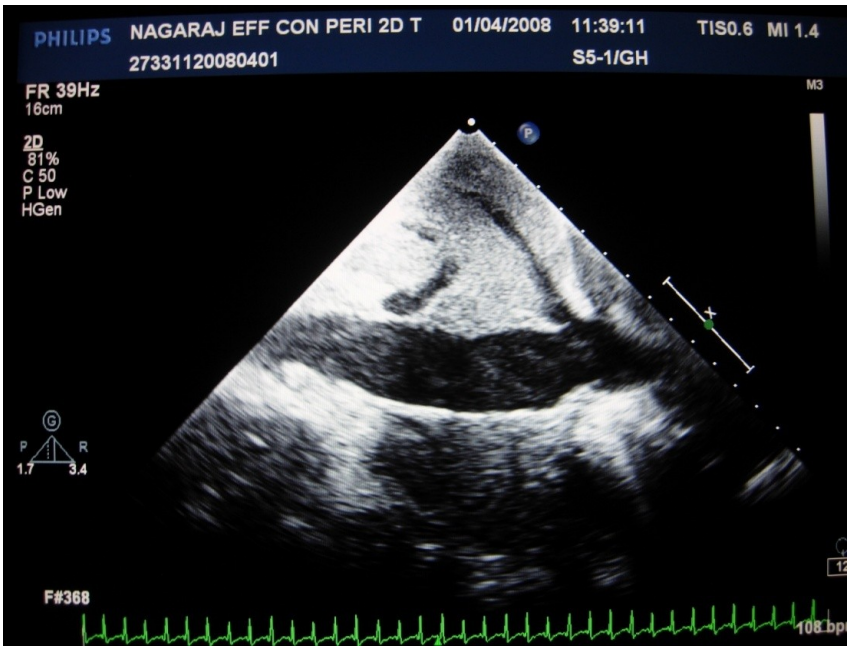

Figure 32 Dilated IVC with intense SEC

Tissue Doppler Imaging (TDI) (Table-22)

Analysis of mitral annular TDI

The mean value of peak early diastolic mitral annular velocity (E') was 12.8 cm/s, which is raised above the normal value of 10 cm/s.

The mean value of peak late diastolic mitral annular velocity (A') was 6.37 cm/s.

The mean value of peak systolic mitral annular velocity (S') was 7.2 cm/s (normal range: 7 to 7.5 cm/s) and was suggestive of normal LV systolic function.

The mean value of ratio (E/E' ratio) between peak early diastolic mitral inflow velocity (E) and peak early diastolic mitral annular velocity (E') was 5.85 (range: 3.29 to 11.17).

Table 22 ***Tissue Doppler Imaging (TDI)***

	Mitral Annulus(cm/s)				Tricuspid Annulus(cm/s)			
Name	S’	E’	A’	E/E’	S’	E’	A’	E/E’
1	7.1	11.7	8.6	38.5/11.7 =3.29	7.8	11.2	6.4	36.8/11.2=3.2
2	6.4	12.8	6.6	58.8/12.8=4.6	7.6	10.6	7.0	55.1/10.6=5.2
3	6.6	13.2	5.5	54.5/13.2 =4.12	7.3	9.6	6.6	39.1/9.6 =4.07
4	7.4	17.4	5.5	100.1/17.4=5.75	8.5	15	6.1	86.6/15=5.77
5	5.3	7.4	5.6	82.7/7.4 =11.17	10.3	14	7.4	95.2/14=6.8
6	6.6	9.9	5.4	65.1/9.9 =6.57	9.8	18.6	8.8	57.7/18.6=3.10
7	7.9	14.1	4.3	97.5/14.1 =6.91	12.9	18.7	3.7	81.4/18.7=4.35
8	9.7	15.1	6.6	58.6/15.1 =3.88	11.2	16.4	4.8	80.6/16.4=4.92
9	6.68	19.5	9.47	81/19.5=4.15	9.6	10.7	5.2	43.8/10.7=4.1
10	7.12	8.87	5.36	67.5/8.87=7.6	8.77	6.24	14.7	47.2/6.24=7.5
11	7.9	11.4	6.2	90.8/11.4=7.96	10.9	17.1	16.4	48.4/17.1=2.83
12	7.8	12.5	7.4	53.6/12.5 =4.28	7.7	13.8	7.8	65.6/13.8=4.76

Analysis of Tricuspid annular TDI

The mean value of peak early diastolic Tricuspid annular velocity (E’) was 13.49 cm/s.
The mean value of peak late diastolic Tricuspid annular velocity (A’) was 7.9 cm/s.
The mean value of peak systolic Tricuspid annular velocity (S’) 9.36 cm/s normal value of S’ > 11.5 cm/s).
Mean value of ratio (E/E’ ratio) between peak early diastolic Tricuspid inflow velocity (E) and peak early diastolic Tricuspid annular velocity (E’) was 4.71 (range: 2.83 to 7.5).

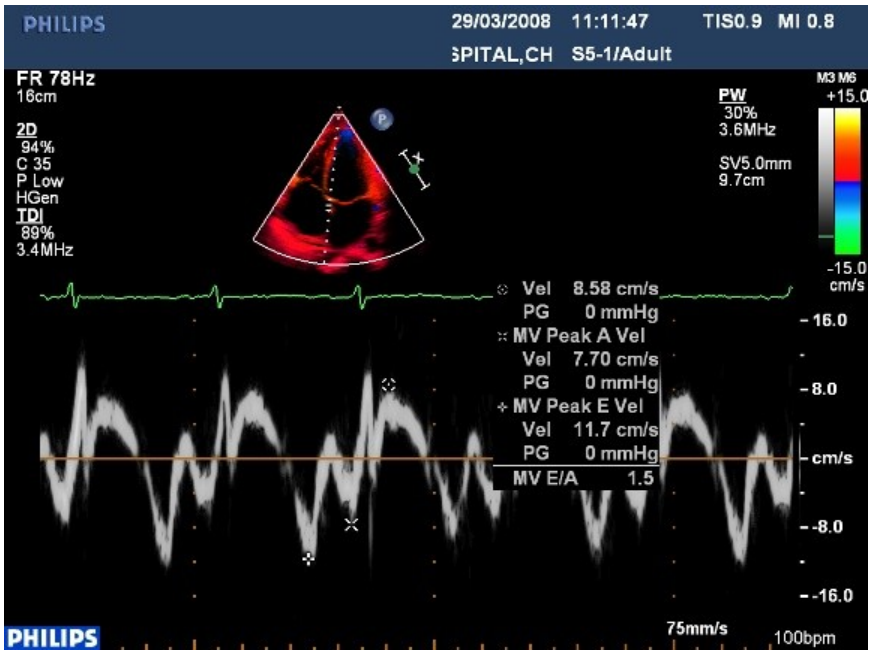

Figure 33 Peak early diastolic mitral annular velocity (E') – Increased in constrictive pericarditis

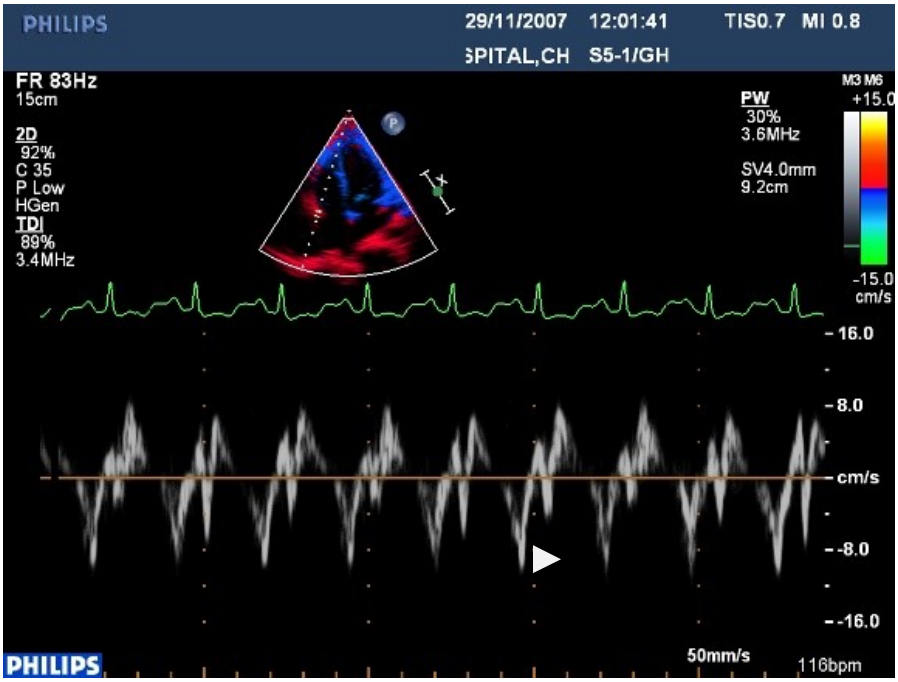

Figure 34 Peak early diastolic tricuspid annular velocity (E') – Increased in constrictive pericarditis

TransEsophageal Echocardiographic (TEE) data analysis (Table-23)

Table 23 ***TransEsophageal Echocardiography***

	Pericardium						
Name	*Thick (mm)*	*Calcific*	*Distribution*	*Pericardial strands*	*Organized PEF*	*SEC*	*RA/RAA Thrombus*
1	LVFW9.7 RVFW6.3	RV	LV >RV	−	−	RA	−
2	LVFW 11 RVFW7	RV	RV>LV	−	RA/ R –AV groove	−	−
3	LVFW 16 RVFW 11 RA 33.6	−	RA>RV>LV	+	+	RA/RV	−
4	LVFW 9.1 RVFW20	Rt AV groove/ RVFW	RV>LV	−	RV>LV	RA	RA/RV
5	LVFW 11 RVFW7 RA 20	−	RA>LV>RV	+	+	−	−
6	LVFW 29.4	−	LV>RV	++	+	RA	−
7	LVFW 11.6 RVFW8.1	?LVFW	LV>RV	−	+	−	−
8	LVFW 11 RVFW7 RA 19	−	RA>LV>RV	−	−	RA/RV	−
9	LVFW 12 RVFW14	Rt AV Groove	RA/RV>LV	−	+	RA/	RAA
10	LVFW 39 RVFW35	−	global	++	+	RA/RV	−
11	LVFW 9.0 RVFW6.6	−	global	−	−	−	−
12	LVFW10 RVFW6.4	−	LV >RV	−	−	−	−

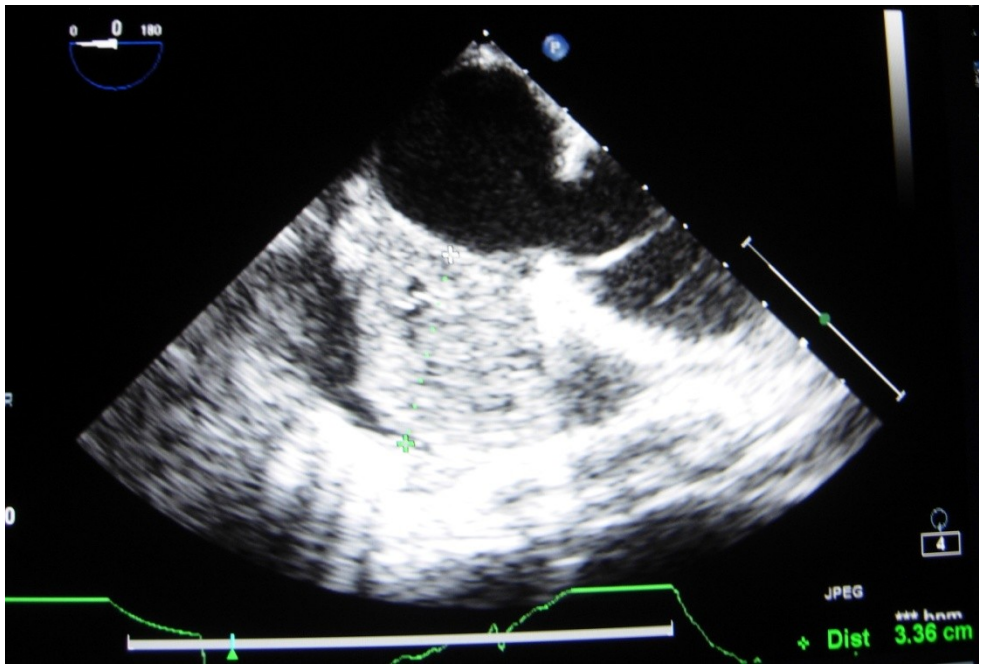

Figure 35 Marked thickening of the pericardium around the Right Atrium shown by TEE

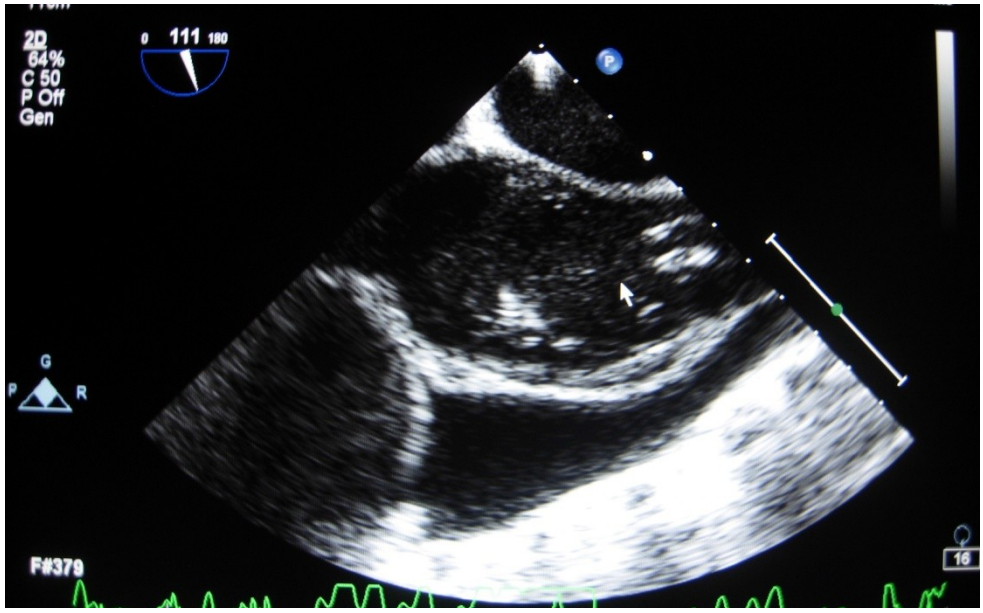

Figure 36 TEE showing intense SEC and thrombus inside the Right Atrium

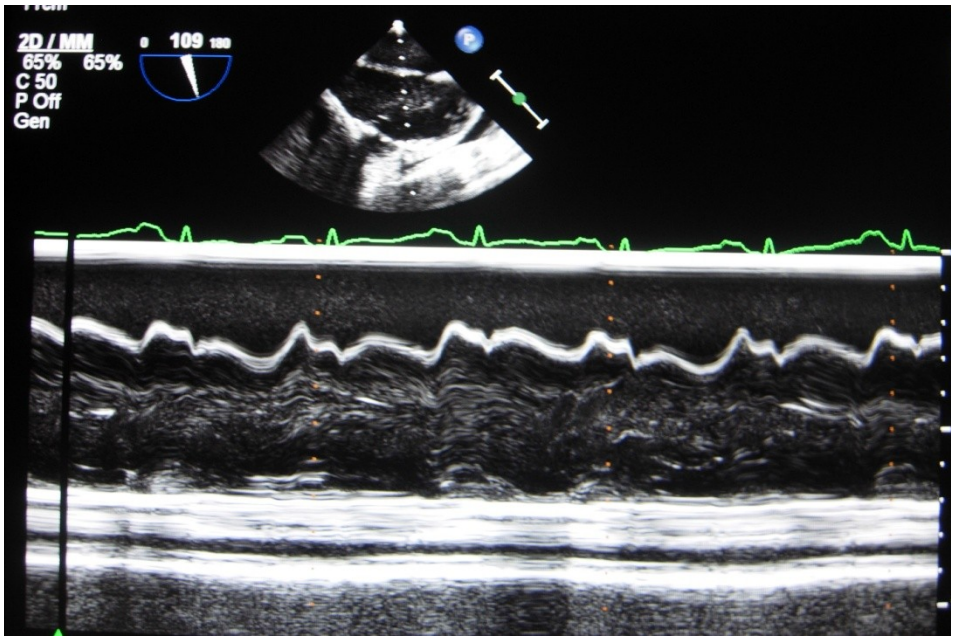

Figure 37 Exaggerated motion of Inter atrial Septum in Constrictive Pericarditis

TEE demonstrated pericardial thickening in all patients. The pericardial thickness was not uniform over the cardiac chambers and varied in thickness over different sites.
The mean pericardial thickness over RV was 10.7 mm (range: 6.3 to 35 mm) and 13.9 mm over LV (range: 9.1 to 39mm).
The pericardium over the RA was thickened in all cases and was the site of maximum involvement in 3 patients (25%). The maximum thickness over RA was 33.6 mm (range: 19 to 33.6 mm)
The distribution of pericardial thickening was as follows – LV > RV in
4 patients (33.3%): RA/RV>LV in 6 patients (50%) and global in 2 patients (16.6%).
Prominent involvement of the pericardium over RA was present in
4 patients.
Calcification was evident by TEE in 5 patients (41.6%). Right AV groove was involved in 3 patients (33.3%). Isolated pericardial calcification over the LVFW and apex was present in one patient (8.3%) and the remaining two patients (16.6%) had predominant involvement of the pericardium over the RV.
Organized pericardial effusion was present in two third of the patients (66.6%) with moderate pericardial effusion in 2 patients (16.6%). Pericardial strands were seen in one third of the cases (33.3%) and were very prominent in 2 cases.
Spontaneous echo contrast (SEC) was present inside the RA in 7 patients (58.3%) along with RV SEC in 3 patients.
Thrombus was present in 2 patients (16.6%): RA thrombus in one patient and RAA thrombus in another patient.

Analysis of Hemodynamic Data (Table-24)

The mean right atrial pressure (mRAP) ranged from 16 to 24 mm Hg (mean value =20 mmHg).
The mean value of peak Right Ventricular systolic pressure(RVSP) was 36.2 mmHg(range: 30 to 50).
The mean value of Right Ventricular end diastolic pressure(RVEDP) was 20.5 mmHg(range: 18 to 24).
The mean value of peak pulmonary artery systolic pressure (PASP) was 34 mmHg (range: 26 to 48).
The mean pulmonary artery pressure (m PAP) ranged from 16 to 36 mm Hg (mean value=25.8).
Mean pulmonary capillary wedge pressure (mPCWP) ranged from 16 to 24 (mean value = 20.5).
The mean value of peak left ventricular systolic pressure(LVSP) was 102.2 mm Hg(range:86 to 129).
Mean left ventricular end diastolic pressure(LVEDP) was 20.25 mm Hg(range 18 to 26).
The early diastolic rapid filling wave (RFW) ranged from 7 to 17 for RV and 5 to 22 for LV.

Table 24 ***Hemodynamic data of patients with constrictive pericarditis***

Name	*Mean RAP*	*Peak RVSP*	*RV RFW*	*Ins↓ RAP*	*RV EDP*	*Peak PASP*	*Mean PAP*	*Mean PCWP*	*Peak LVSP*	*LV RFW*	*LV EDP*	*Mean Ao Pressure*
1	16	33	8	−	19	28	20	20	100	10	18	90
2	18	40	17	−	18	40	30	22	120	20	20	100
3	18	30	8	−	18	28	22	20	86	8	21	70
4	21	30	12	−	18	26	16	16	90	20	21	70
5	20	32	7	+	22	30	24	20	96	5	21	70
6	24	36	8	−	20	34	28	20	120	12	18	100
7	20	40	9	−	24	36	28	24	129	22	26	80
8	24	50	8	−	24	48	36	22	96	10	20	80
9	20	38	10	−	20	32	26	21	86	11	18	70
10	22	36	9	−	20	34	24	20	100	12	21	70
11	21	40	10	−	22	40	30	22	110	8	20	74
12	16	30	8	−	21	32	26	19	94	7	19	70

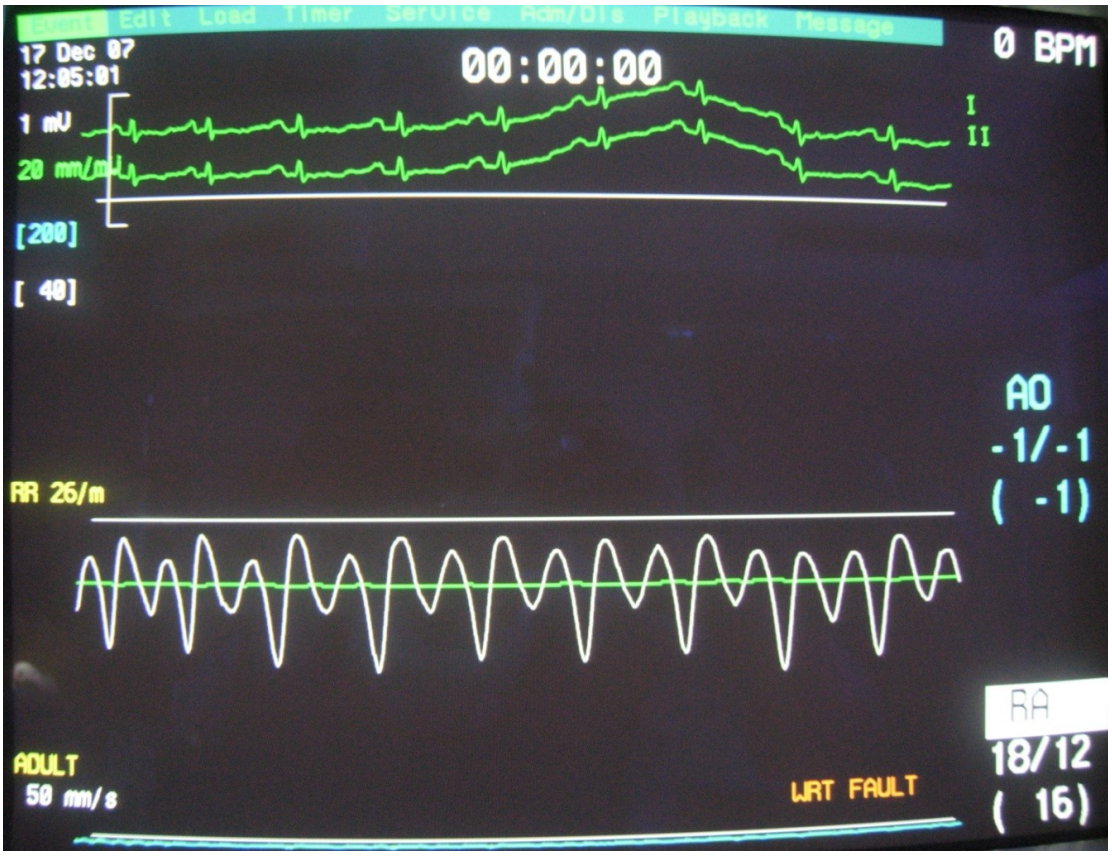

Figure 38 RA Pressure tracing –demonstrating typical M or W pattern and absence of respiratory variation in mean pressure. (Kussmaul's sign)

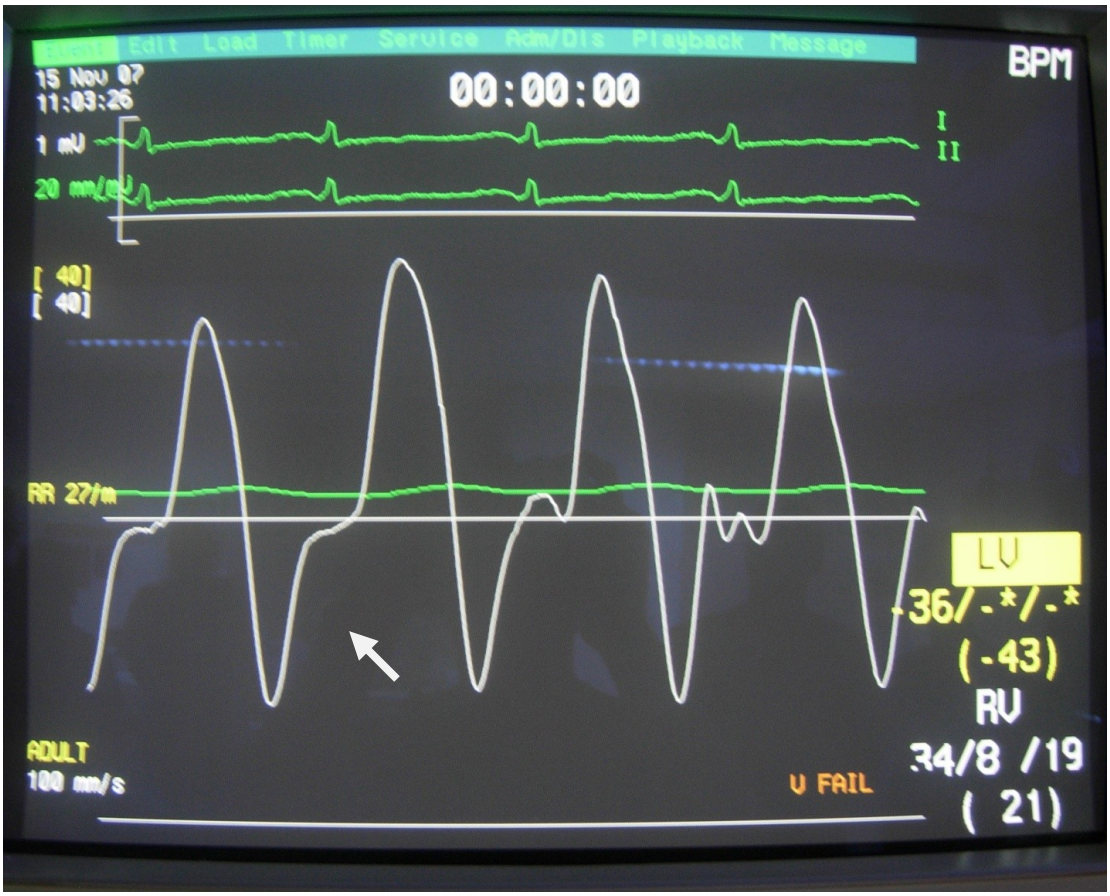

Figure 39 Right ventricular pressure tracing showing early diastolic dip and rapid filling and followed by a plateau (Square root sign)

Figure 40 Increase in gradient between PCWP and early diastolic LV pressure during expiration(arrow head) compared with inspiration(arrow)

Figure 41 A

Figure 41B

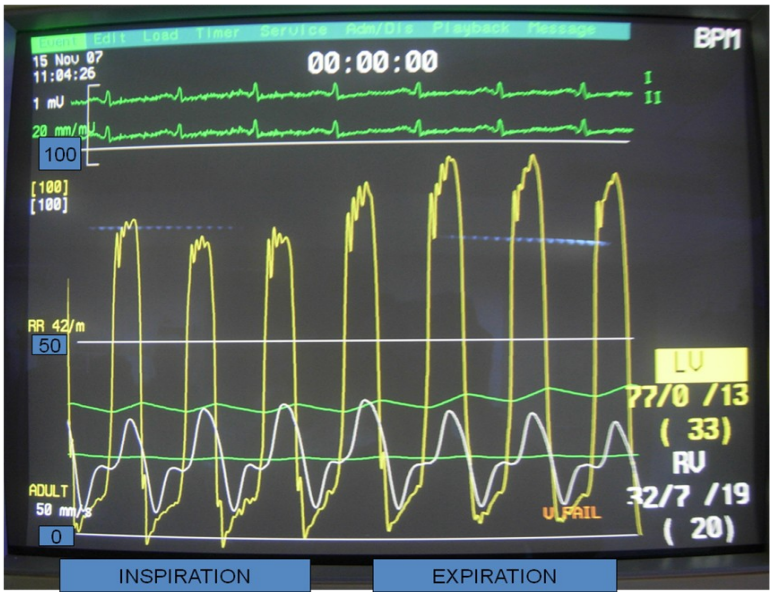

Figure 41 A, B Simultaneous recording of RV and LV pressure tracings showing Ventricular discordance due to interdependence - Inspiratory increase in RVSP &decrease in LVSP and Expiratory increase in LVSP & decrease in RVSP

Analysis of Hemodynamic Data for Conventional and Dynamic Catheterization Criteria (Table-24.25)

The diastolic pressures of all the four chambers were almost equal with < 5mm Hg difference and were around 20 mmHg in all cases.

Also the difference between LVEDP and RVEDP was < 5 mm Hg with a mean value of 2.25 mm Hg(range: 1 to 4 mm Hg) was present in all cases.

RVEDP was more than one third of the Right ventricular systolic pressure in all patients.

The pulmonary artery systolic pressure was < 55 mm Hg in all cases.

The early diastolic rapid filling wave ≥ 7 mm Hg was present in 11 patients in LV (91.6%) and was invariably present in all patients in RV.

Respiratory variation in mRAP ≤ 3mm Hg (Kussmaul's sign) was demonstrable in 11 patients (91.6%)

Dynamic respiratory criteria for constrictive pericarditis

Equal or more than 5 mm Hg increase in the gradient between PCWP and LV early diastolic pressure during expiration compared with inspiration was present in 11 patients (91.6%).

Ventricular discordance due to ventricular interdependence – inspiratory increase in RVSP and decrease in LVSP and opposite changes during expiration was demonstrable in all the 12 patients (100%).

Table 25 Analysis of Hemodynamic Data for Conventional and Dynamic Catheterization Criteria

Name	*Square root sign*	*LVEDP-RVEDP (<5mmHg)*	*RVEDP>1/3 RVSP*	*PASP <55mm Hg*	*Insp↓ MRAP*	*VID*	*PCWP-LVEDP> 5mm Hg*
1	RV&LV	2	+	+	absent	+	+
2	RV&LV	2	+	+	absent	+	+
3	RV&LV	3	+	+	absent	+	+
4	RV&LV	3	+	+	absent	+	+
5	RV	1	+	+	present	+	+
6	RV&LV	4	+	+	absent	+	+
7	RV&LV	2	+	+	absent	+	+
8	RV&LV	4	+	+	absent	+	+
9	RV&LV	2	+	+	absent	+	+
10	RV&LV	1	+	+	absent	+	+
11	RV&LV	2	+	+	absent	+	+
12	RV&LV	2	+	+	absent	+	+

Table 26 Hemodynamic and TDI correlation in patients with Constrictive Pericarditis (Mitral Annulus)

Name	***PCWP (Cath Data)***	***E/E' (TDI Data)***	
		Mitral	***Tricuspid***
1	**20**	**3.29**	**3.2**
2	**22**	**4.6**	**5.2**
3	**20**	**4.12**	**4.07**
4	**16**	**5.75**	**5.77**
5	**20**	**11.17**	**6.8**
6	**20**	**6.57**	**3.10**
7	**24**	**6.91**	**4.35**
8	**22**	**3.88**	**4.92**
9	**21**	**4.15**	**4.1**
10	**20**	**5.36**	**7.5**
11	**22**	**7.96**	**2.83**
12	**19**	**4.28**	**4.76**

Table 27 Hemodynamic and TDI correlation in patients with Constrictive Pericarditis – Tricuspid Annular TDI& Mean RAP

Name	*Cath Data* *mRAP(mm Hg)*	*TDI Data* *E/E'*
1	**16**	**3.2**
2	**18**	**5.2**
3	**18**	**4.07**
4	**21**	**5.77**
5	**20**	**6.8**
6	**24**	**3.10**
7	**20**	**4.35**
8	**24**	**4.9**
9	**20**	**4.1**
10	**22**	**7.5**
11	**21**	**2.8**
12	**16**	**4.76**

PERICARDIECTOMY - INTRAOPERATIVE PICTURES

Figure 42 A

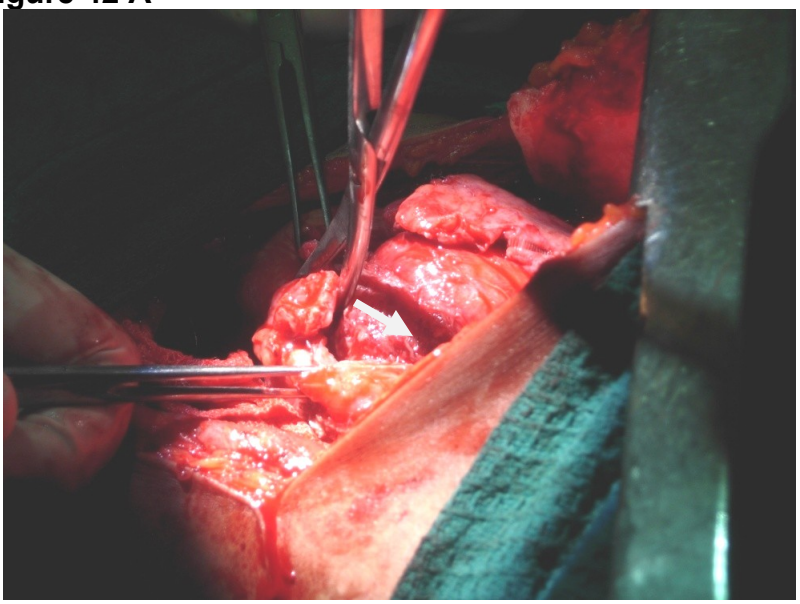

Figure 42 B

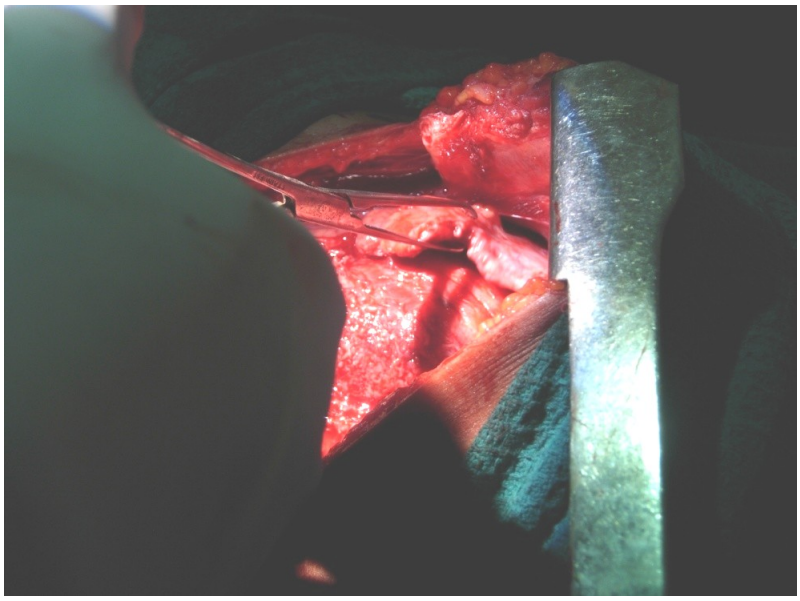

Figure 42 A, B Markedly thickened parietal pericardium

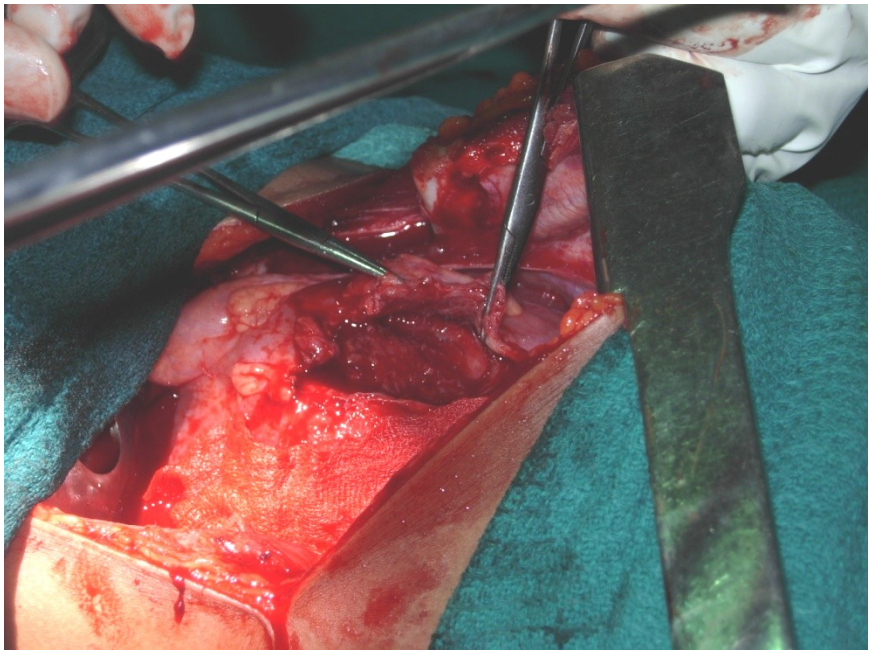

Figure 43 Thickened epicardial layer

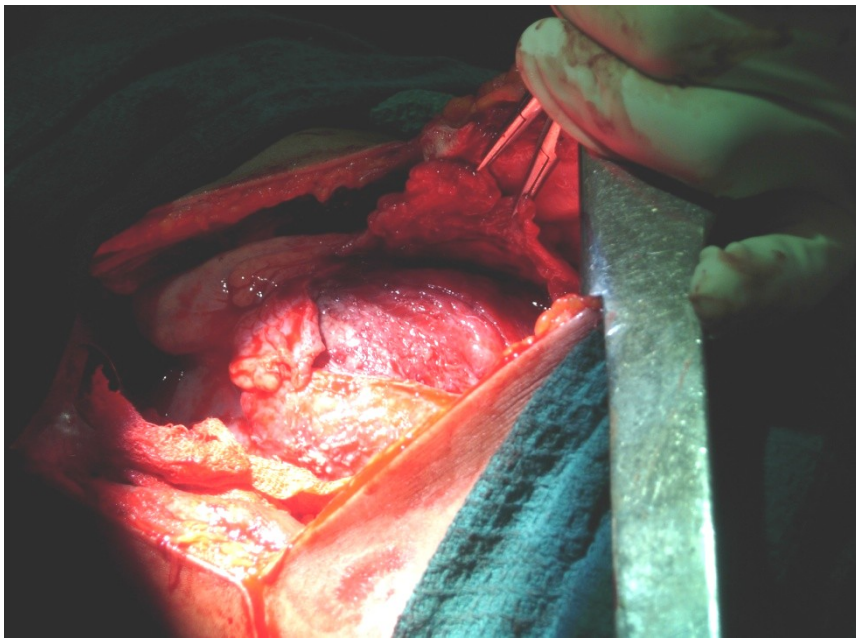

Figure 44 Excision of the thickened epicardium exposes the underlying myocardium Post pericardiectomy Echocardiography

Figure 45 A

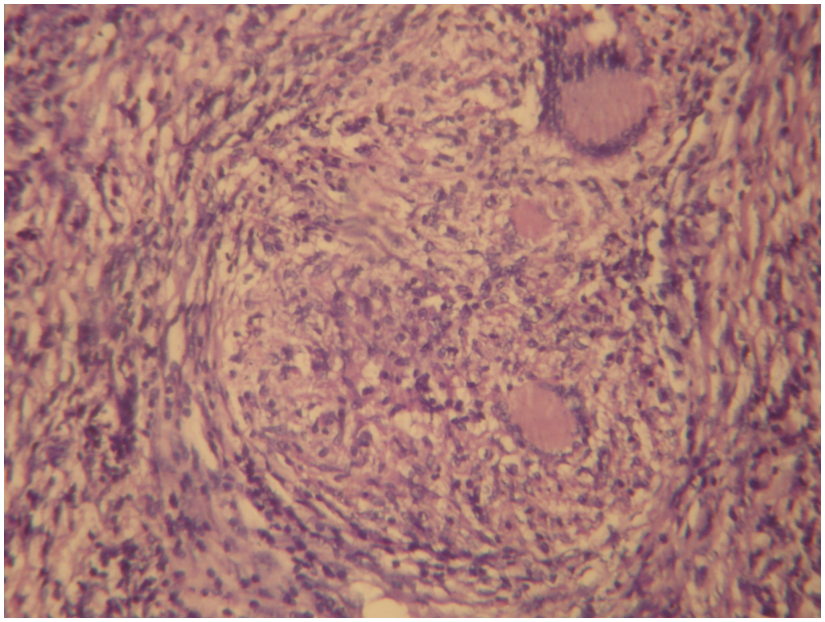

Figure 45 B

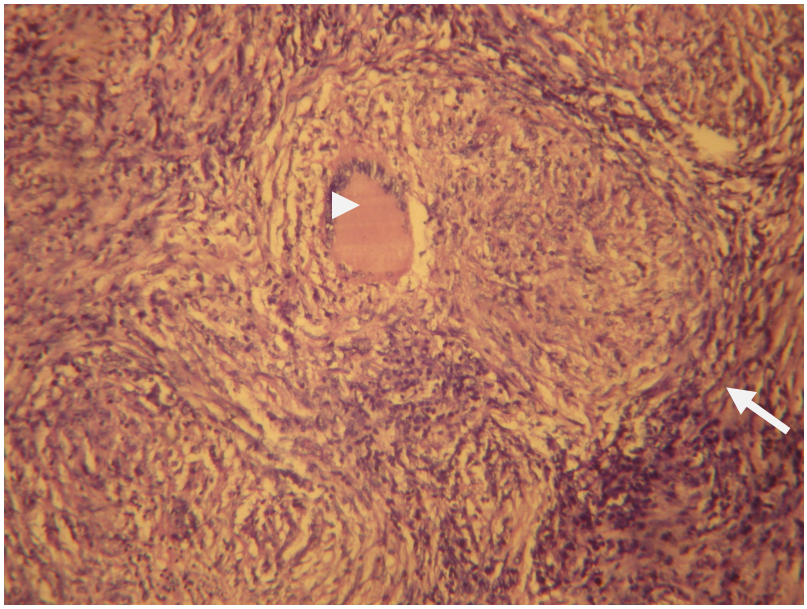

Figure 45 A,B photomicrographs of a patient with tuberculous constrictive pericarditis showing tuberculous granuloma(arrow) with multinucleate giant cells(arrow head)

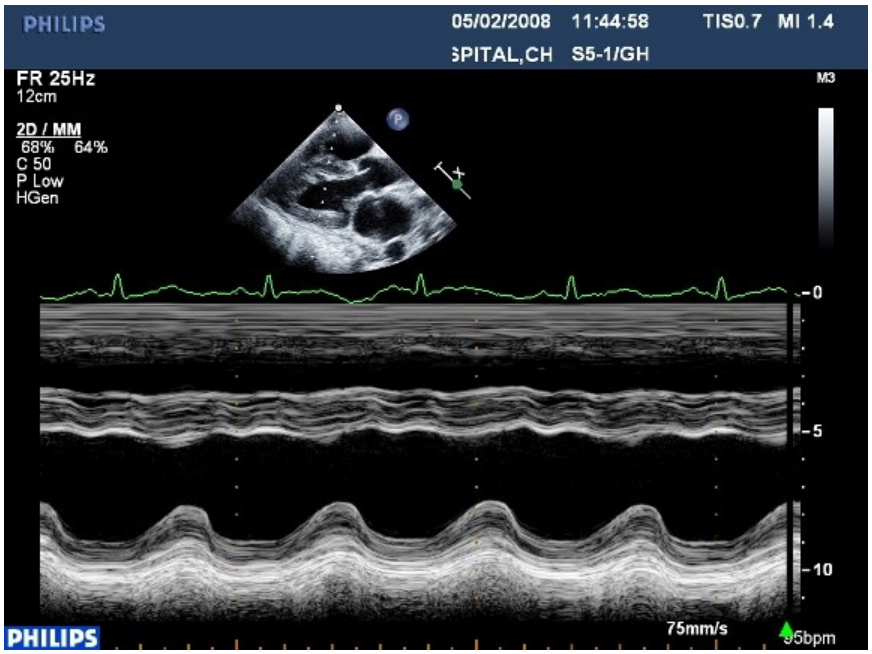

Figure 46 M Mode Echo showing normalization of LVPW relaxation during diastole

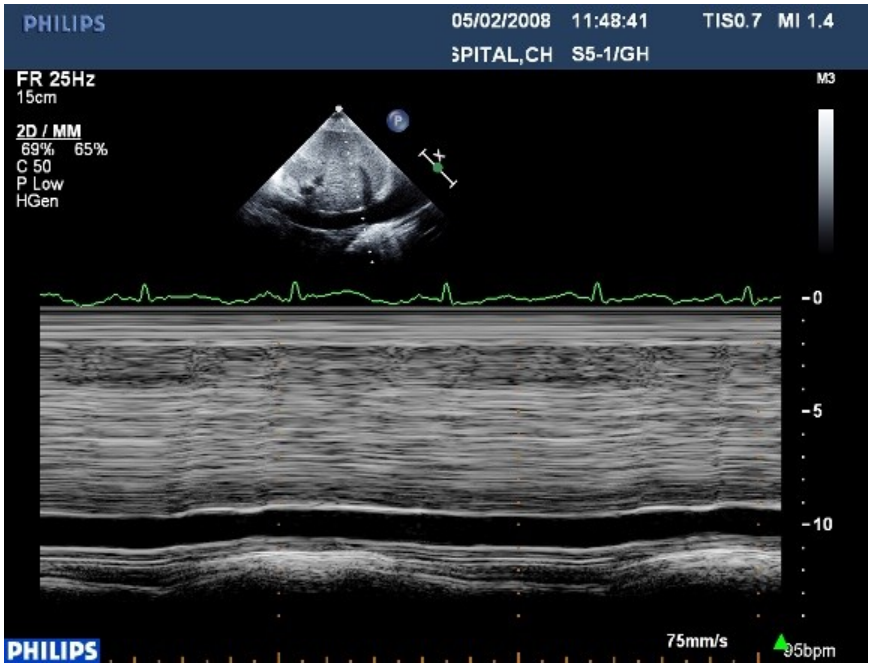

Figure 47 IVC (M Mode echo) is normal and non dilated with significant Respiratory variation in size

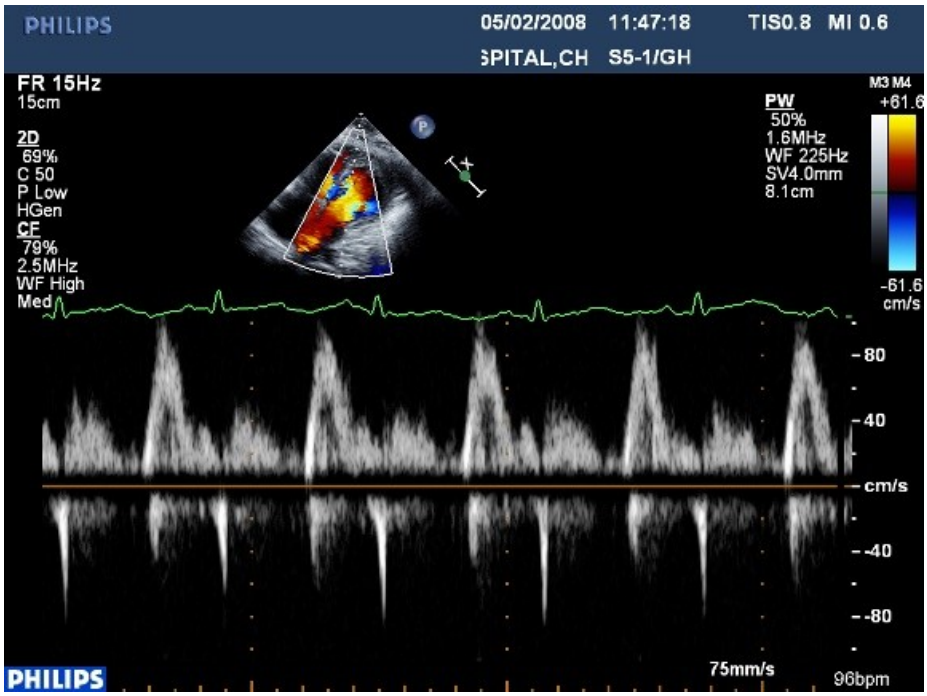

Figure 48 Mitral early diastolic peak velocity (E) showing no significant respiratory variation

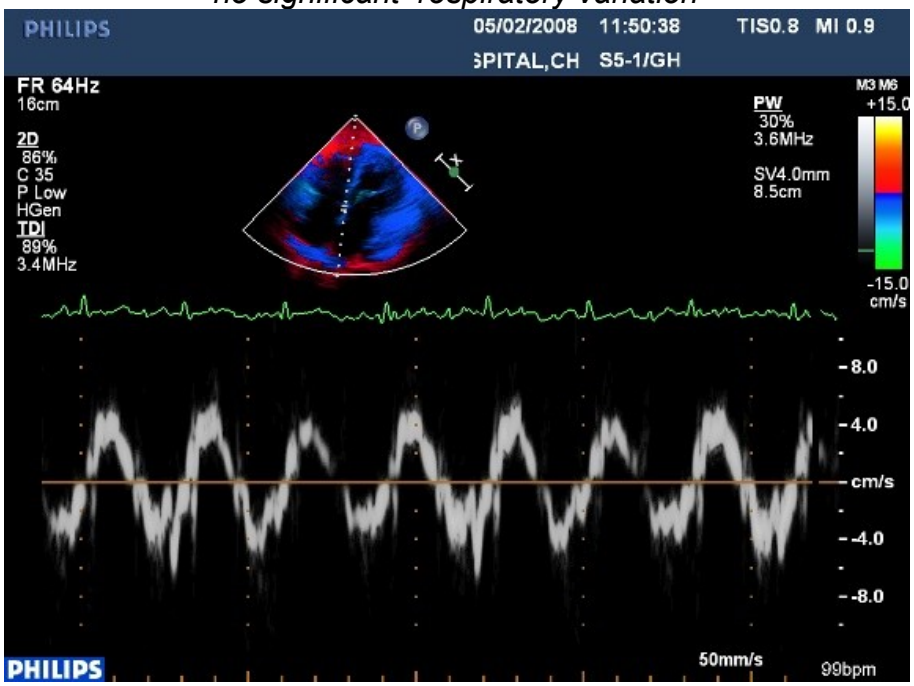

Figure 49 Peak early diastolic mitral annular velocity (E') – showing normalized velocity following pericardiectomy with reversal annulus paradoxus

DISCUSSION

It is important to make the diagnosis of constrictive pericarditis because untreated patients have progressive hemodynamic and physical deterioration and a limited life span. Conversely, complete resection of all constrictive pericardium can result in marked improvement in symptoms as well as prolongation of life with possible cure.

Of the 12 patients of the study group, the clinical and echocardiographic features of constrictive pericarditis had excellent correlation with CT scan and cardiac catheterization studies.

All these patients are younger (<50 years) and most of them presented relatively earlier (median duration of 5 months) than the patients in the previous studies. This is due to higher prevalence of subacute form of constrictive pericarditis than the classic chronic calcific constrictive pericarditis which was the dominant form of the disease till few years ago. Also this early presentation can be ascribed to improved diagnostic modalities, especially wide spread availability of echocardiography.

In addition to the earlier presentation, the other interesting features in this study are Dyspnoea with abdominal distention which was invariable in all patients.

Ascites precox which is a characteristic feature of constrictive pericarditis was present in most of the patients (91.3%).

History of fever and chest pain suggestive of pericarditis with or without effusion at the onset of illness was present in 50% of the cases. This is in contrast to the previous studies in which an episode of pericarditis at onset of constriction process was uncommon. This underscores the need for adequate evaluation and follows up of patients with features of pericarditis.

Since such a feature is a common characteristic of tuberculous pericarditis, this explains the common occurrence in our clinical setting where tuberculosis is still the common cause of constrictive pericarditis.

In contrast, tuberculosis has become rare, and dominated by prior cardiac surgery and mediastinal irradiation in the Western population. Also none of the patients in this study had prior cardiac surgery, mediastinal irradiation, uremia or malignancy as a cause for constrictive pericarditis.

History of pericardiocentesis along with anti TB treatment was present in 2 patients in this study. This is compatible with the pathophysiology of constrictive pericarditis, where the disease is the sequelae of healing process and anti TB treatment does not always prevent the development of constrictive pericarditis.

Elevated jugular venous pressure, hallmark of constrictive pericarditis was present in 11 out of 12 patients. Its absence one patient could be related to the prior diuretic therapy.

The typical pattern of jugular venous wave form(y>x) was present in three fourth of the patients. Two patients had equally prominent x and y waves and moderate pericardial effusion demonstrated subsequently during echocardiography, consistent with the diagnosis of Effusive - constrictive pericarditis.

Kussmaul's sign was present in 10 patients (84%).Pulsus Paradoxus was present in 3 patients (25%) among which, two patients had features of Effusive - constrictive pericarditis.

The characteristic auscultatory sign of constrictive pericarditis- early diastolic

pericardial knock was audible in three fourth of the patients. This is higher than in the Western studies. Systolic murmur of mitral regurgitation was audible in one fourth of the patients, although mitral regurgitation was demonstrable in half of the patients during echocardiography (mild MR-5 patients; moderate MR -1 patient).There was no audible murmur of tricuspid regurgitation or pericardial rub.

All the patients in the study were in the sinus rhythm. Although atrial fibrillation has been documented in up to 50% of the Western patients, it was notably absent in this study. Absence of atrial fibrillation is possibly due to relatively shorter duration of symptoms and predominantly subs acute nature of the disease, although in one patient the duration of illness was 24 months, but persisted in sinus rhythm.

Sinus tachycardia was common (83.3%), though not invariable in this study. The ECG was abnormal in all the patients. The electrocardiographic features such as abnormal notched P waves, low voltage QRS complexes, non specific ST-T changes were common.

Heart size was normal in one third of the patients and mild to moderate cardiomegaly was present in the remaining patients. There was no gross cardiomegaly.

Left atrial enlargement in chest radiography was uncommon and evident in 2 patients only (16.6%).Pulmonary venous congestion was present in two third of the patients and distention of SVC in 83% of cases. Frank pulmonary was not a feature.

Thick shaggy calcification of pericardium in chest radiography, suggestive of chronic calcific constrictive pericarditis was present in only in 2 patients (16.6%) which is in concordance with other studies. This suggests that this classic sign has become an uncommon entity.

But fluoroscopic screening revealed pericardial calcification in 4 patients (33.3%) Pericardial calcification was seen in 3 patients (25%)by transthoracic echocardiography and in 5 patients(50%) by TransEsophageal echocardiography. Also TEE was more sensitive to diagnose AV groove calcification which was present in 3 patients(25%). But CT scan demonstrated pericardial calcification in one third of the patients who also had pericardial calcification in fluoroscopy. Similar to TEE, AV groove calcification was present in 3 patients (25%).

M-Mode echocardiography was insensitive for confirming the presence or absence of pericardial calcification(2 patients had calcification).

Pericardial thickening was demonstrable by M mode echocardiography in all the patients. The mean thickness was 16.1 mm posteriorly. Anterior pericardial thickening was seen in 3 patients (range: 3.5 to11 mm)

2D echocardiography could not demonstrate pericardial thickening in one patient which was evident in TEE and CT scan. This patient had the minimal thickening of all- 4 mm by CT scan and 6.3 mm by TEE. Thus TEE and CT scan are more sensitive than 2D echocardiography to demonstrate pericardial thickening.

The mean pericardial thickness was 15 mm (range: 4 to35 mm)by CT scan and 13.9 mm (LV);10.7 mm(RV).Pericardial thickness was maximal over RA in one third of the cases. RA and RV were involved more frequently and severely than LV

Pericardial effusion was present in two third of the patients by TEE and CT scan .Prominent pericardial strands were better visualized by TEE. Prominent pericardial strands are usually associated with a risk of future development of constriction, if not developed earlier.

CT scan did not show any intracardiac thrombus in this study. Interestingly, TEE showed presence of thrombus in two patients- (16.6%). One patient had thrombus inside the Right atrial appendage and in another patient thrombus was inside the RA-IVC junction. Spontaneous echo contrast (SEC) better seen by TEE and was present inside the RA in 7 patients. In addition RV SEC was associated in 3 cases.

This high incidence of SEC and intracardiac thrombus are secondary to venous stasis with raised venous pressure. The intracardiac thrombus is a potential source of pulmonary embolism with significant morbidity and mortality.

Thus TEE is very essential in the evaluation of patients with constrictive pericarditis. It provides excellent data on pericardial thickening and calcification, organized/loculated effusion. TEE is as sensitive as CT scan for pericardial thickening. In addition it provides valuable information regarding pericardial strands and intracardiac thrombi which are important during pericardiectomy.

Also TEE provides better Doppler evaluation of pulmonary venous flow and involvement of the AV groove by the constricting pericardium.

Echocardiographic features of constrictive pericarditis

Septal bounce, diastolic septal notches, dilated IVC with <50% respiratory variation(IVC plethora) were consistently present in all the patients .Thus the sensitivity of these findings is higher(almost 100%) when taken together in an appropriate clinical settings.

Respiratory variation in the Mitral early diastolic peak velocity (E) of >25% increase during expiration compared to inspiration was present in 10 patients(88.3%).

Respiratory variation in the Tricuspid early diastolic peak velocity (E) of >40% increase during inspiration compared to expiration was present in 8 patients (66.6%).

Ratio(S/D) between Pulmonary venous systolic flow (S) velocity and Pulmonary venous diastolic flow (D) velocity was <1.0 in all patients. It was >0.65 in 10 patients (83.3%) and one patient had value of <0.50. This Doppler finding differentiates constrictive pericarditis from restrictive cardiomyopathy where the S/D ratio is <0.50 and characterized by the absence of respiratory variation.

The respiratory variation in Pulmonary venous diastolic flow (D) velocity of >40% increase during expiration was present two patients only (16.6%).

Hepatic Venous diastolic flow reversal (DR) analysis during inspiration and expiration revealed an exaggerated expiratory increase of diastolic flow reversal (9.7% increase during expiration) in all patients (100%) in this study.

Tissue Doppler Imaging (TDI)

The mean value of peak early diastolic mitral annular velocity (E') was 12.8 cm/s. which is raised above the cut off value of ≥ 8 cm/s in all the patients.

E' was positively correlated with PCWP (r=0.69, P=0.027) and LVEDP (r=0.69, P=0.029).

The mean value of ratio (E/E' ratio) between peak early diastolic mitral inflow velocity (E) and peak early diastolic mitral annular velocity (E') was 5.85 (range: 3.29 to 11.17). Consequently, there was a significant inverse correlation between E/E' and PCWP(r=−0.63, P=0.004) and LVEDP (r=−0.60, P=0.004). Despite high LV filling pressures, E/E' was<15 in all patients.

Post operatively repeat TDI showed decrease in E' value (mean 7.4 cm/s) and E/E' ratio was in the range of 7.6 to 9.4.

Analysis of Hemodynamic Data

The following conventional and hemodynamic criteria were present in > 90% of the patients in this study. The most sensitive and specific hemodynamic criteria of constrictive pericarditis- Ventricular discordance due to ventricular interdependence was demonstrable invariably in all patients.(100%)
The diastolic pressures of all the four chambers were almost equal with <5mm Hg difference and were around 20 mmHg in all cases.
Also the difference between LVEDP and RVEDP was < 5 mm Hg with a mean value of 2.25 mm Hg was present in all cases.
RVEDP was more than one third of the Right ventricular systolic pressure in all patients.
The pulmonary artery systolic pressure was < 55 mm Hg in all cases.
The early diastolic rapid filling wave ≥ 7 mm Hg was present in 11 patients in LV (91.6%) and was invariably present in all patients in RV.
Respiratory variation in mRAP ≤3mm Hg (Kussmaul's sign) was demonstrable in 11 patients (91.6%).
Equal or more than 5 mm Hg increase in the gradient between PCWP and LV early diastolic pressure during expiration compared with inspiration was present in 11 patients (91.6%).
Ventricular discordance due to ventricular interdependence – inspiratory increase in RVSP and decrease in LVSP and opposite changes during expiration was demonstrable in all the 12 patients (100%).

Hemodynamic and Tissue Doppler Correlates in Constrictive Pericarditis

Previously, the hemodynamic diagnosis of constrictive pericarditis has focused on pressure variables measured during held respiration. However, suboptimal specificity and sensitivity of these criteria have limited the clinical applicability of these criteria in individual patients.
Hatle et al provided insight into the dynamic respiratory changes in ventricular filling and pressures that occur in patients with constrictive pericarditis. These changes include a dissociation of intrathoracic and intracardiac pressures and increased ventricular interaction. In normal individuals and cardiac patients without constrictive pericarditis, there is an inspiratory decrease in intrathoracic pressures that is transmitted to the cardiac chambers. Thus, transmitral filling pressure during early diastole is essentially unchanged throughout the respiratory cycle, and there is minimal change in left ventricular filling. In the presence of a constricting pericardium around the heart, the inspiratory decrease in pulmonary venous and intrathoracic pressure is not transmitted into the cardiac chambers, resulting in a reduced transmitral pressure gradient and less ventricular filling. The constricting pericardium also results in an increase in ventricular interaction, so that as the left ventricular volume decreases, there is a corresponding increase in right ventricular volume.
These pathophysiological abnormalities have been applied to Doppler echocardiographic studies in which the dissociation of intrathoracic and intracardiac pressures is manifested by an inspiratory increase in the peak tricuspid flow velocity and a simultaneous decrease in mitral flow velocity, with opposite changes occurring in expiration.
Although these Doppler findings are usually diagnostic in the presence of other clinical and noninvasive findings consistent with constrictive pericarditis, both false-positive and false-negative results exist. Severe lung disease with marked respiratory changes in

intrathoracic pressures and movement of the sample volume relative to the heart can cause changes in the mitral flow velocity curves mimicking those of constrictive pericarditis. Marked increases in left atrial pressures may mask these Doppler respiratory changes. In addition, in the presence of irregular rhythms, such as atrial fibrillation, it is difficult to determine the significance of changes in the initial mitral velocities.
Thus, there are instances when additional diagnostic methods for evaluating possible constrictive pericarditis are helpful.
In this study, the dynamic respiratory changes in cardiac hemodynamics in patients with constrictive pericarditis were assessed by cardiac catheterization and correlated with the TDI.
The finding of increased ventricular interaction, as assessed by respiratory discordance of left ventricular and right ventricular pressures, was the most reliable hemodynamic factor for distinguishing patients with constrictive pericarditis from those with other disease entities.
In patients with constrictive pericarditis, there was a consistent increase in right ventricular pressure during peak inspiration, a time when left ventricular pressure is lowest. These findings are concordant with prior studies in dogs with experimental cardiac tamponade and confirm the earlier observations of Hatle et al.
The principal finding of this study is the inverse correlation between E/E' and LV filling pressures in patients with CP. This is in contrast to the positive correlation between E/E' and LV filling pressures in patients without a pericardial abnormality. Hence, the term **"annulus paradoxus"** was proposed by Nishimura et al to describe the paradoxical behavior of the mitral annulus in CP.
The assessment of LV filling pressure is clinically important in patients with established heart disease and usually requires invasive hemodynamic measurement. Several noninvasive Doppler echocardiographic indices that use conventional mitral inflow parameters have been proposed to estimate LV filling pressure. Because E is directly influenced by left atrial pressure and inversely altered by changes in the time constant of relaxation, an increase in left atrial pressure can override the effects of impaired relaxation.
Recently, combining E with E' has been proposed as a tool for assessing LV filling pressures. Several investigators have shown that the E/E' ratio is valid for determining LV filling pressures by offsetting the effects of relaxation on E in estimating filling pressures. Nagueh et al validated the relationship between E/E' and PCWP in patients with either impaired or pseudonormal relaxation or with sinus tachycardia. Ommen et al found that an E/E' >15 identified increased LV filling pressure. In addition, they showed that E/E' provided better estimates of LV filling pressures than pulmonary venous flow pattern or the Valsalva maneuver. However, these studies did not include patients with CP.

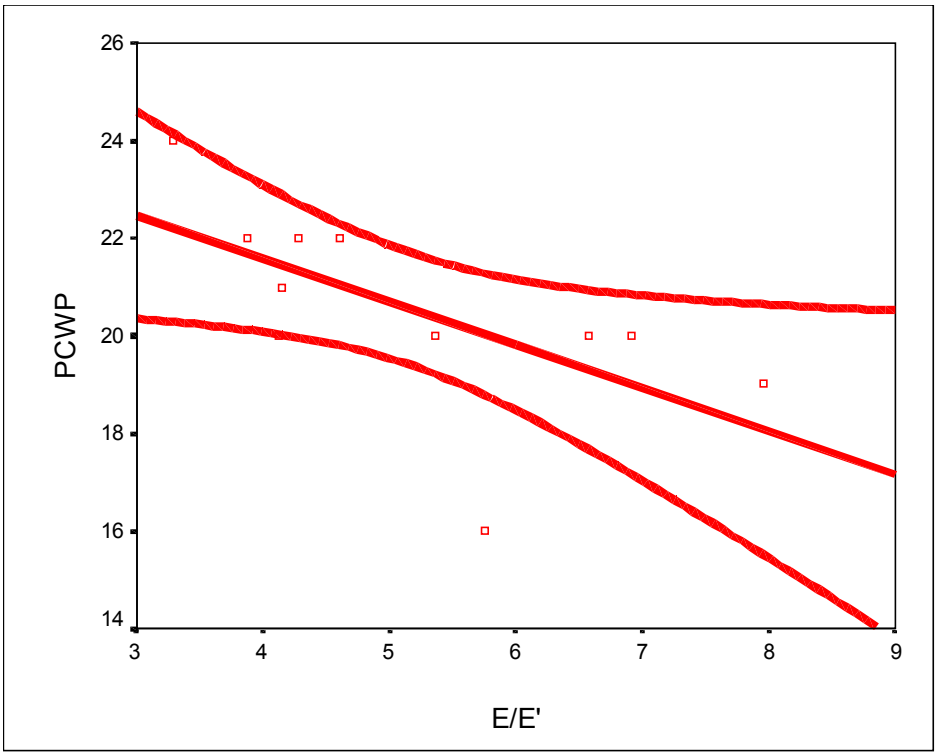

Figure 50 Inverse correlation between PCWP and mitral E/E' ratio in constrictive pericarditis

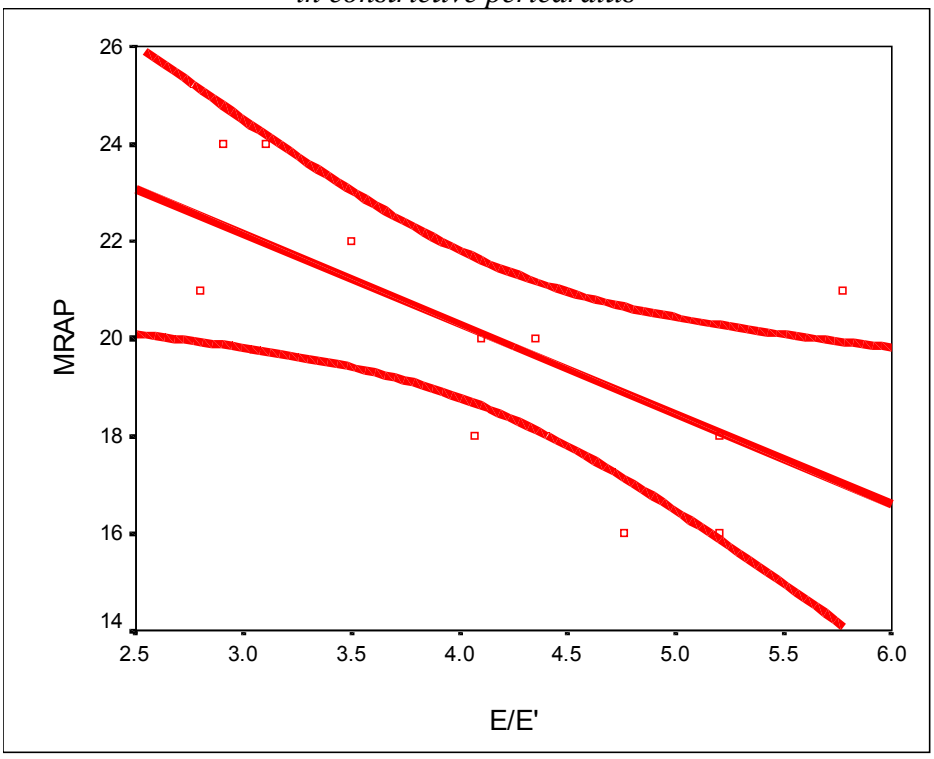

Figure 51 Inverse correlation between mRAP and Tricuspid E/E' ratio in constrictive pericarditis

In CP, E' is usually well preserved or even accentuated, despite increased filling pressures and the finding of preserved E' has been suggested as being clinically useful for

distinguishing CP from restrictive cardiomyopathy.
In this study, a significant inverse correlation was found between E/E' and LV filling pressures in patients with CP, which is the inverse of the correlation shown in patients with a primary myocardial disease. The plausible explanation for this finding is the exaggerated longitudinal motion of the mitral annulus, despite high filling pressures in patients with CP, because the lateral expansion of the entire heart is limited by the constricting pericardium. The more severe constriction with a higher filling pressure, the more accentuated is the longitudinal motion of the mitral annulus. This explanation can be supported by the finding that E' was decreased after pericardiectomy in 3 patients who had a repeat measurement of E' postoperatively. In addition, normal relaxation and a small LV cavity with good systolic function may result in increased longitudinal excursion and E'.
In this study, E' was measured from the septal annulus because its velocity is less influenced by the pericardium. E' from the lateral annulus is usually higher than that of the septal annulus in patients without pericardial abnormality. However, it is potentially affected by the calcification or adhesion of the pericardium in patients with CP.

NEWER AND INTERESTING OBSERVATIONS

Certain newer and interesting findings have been observed from this study.

- Because of the established fact that the inverse correlation between PCWP and mitral E/E' (annulus paradoxus) in constrictive pericarditis , it seems logical to hypothesize possible inverse correlation between mean Right atrial pressure and Tricuspid E/E'[(ratio between peak early diastolic Tricuspid inflow velocity (E) and peak early diastolic Tricuspid annular velocity (E')]
  In this study, the mean tricuspid E/E' was 4.71. (Range: 2.83 to 7.5) and was less than an arbitrary value of 15(Tricuspid E/E' < 15) and inversely correlated with the mean Right atrial pressure (mean value: 20 mm Hg, range: 16 to 24 mmHg). Since RA/RV are involved more commonly and severely than LV in patients with constrictive pericarditis, it is imperative that RV filling pressure and Tricuspid E/ E' depict the inverse correlation similar to PCWP and mitral E/E'. But this new interesting observation needs further study for confirmation.
- The high incidence of RA/RAA thrombus in patients with constrictive pericarditis as highlighted in this study, emphasizes the role of TEE in planning surgical treatment and preventing significant morbidity and mortality from pulmonary embolism.
- Since, significant proportion of the patients with constrictive pericarditis have marked pericardial thickening around RA, total pericardiectomy including the pericardium around RA is mandatory for good surgical results.

This finding can better be evaluated by TEE.

CONCLUSION

Patients with clinically suspected constrictive pericarditis who have classic findings on Doppler echocardiography can now undergo pericardiectomy without the need for cardiac catheterization. Only the patients in whom there have been equivocal findings either on clinical presentation or on noninvasive testing then undergo further hemodynamic assessment.

Echocardiography has replaced invasive cardiac catheterization for various hemodynamic assessments. Of all of the hemodynamic measurements used in daily clinical practice, LV filling pressure is one of the most frequently used. With the advent of TDI and other recent developments in the field echocardiography, it has become a truly versatile and reliable hemodynamic imaging tool. And, echocardiography has become a noninvasive Swan-Ganz catheter.

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SELECTED ABBREVIATIONS AND ACRONYMS

CP	= constrictive pericarditis
CCP	= calcific constrictive pericarditis
TB	= Tuberculosis
LVDP	= left ventricular diastolic pressure
LVEDP	= left ventricular end-diastolic pressure
LVSP	= left ventricular systolic pressure
MRAP	= mean right atrial pressure
PASP	= pulmonary artery systolic pressure
PCWP	= pulmonary capillary wedge pressure
RFW	= rapid filling wave
RVEDP	= right ventricular end-diastolic pressure
RVSP	= right ventricular systolic pressure